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EDITORIAL

BOVINE BRUCELLOSIS

In this issue of the *British Veterinary Journal* we publish an article on bovine brucellosis from the pen of Dr. Axel Thomsen, Copenhagen, whose interest in the subject will be known to most of our readers. Although methods of control of brucellosis are now being satisfactorily practised in many countries, consequent upon the introduction of vaccination, it is refreshing to note that studies on different aspects of the condition are still being pursued. It is shown that the stage of pregnancy at which infection takes place has a considerable bearing upon the incubation period of the disease. It may not have been generally realised that abortion and stage of pregnancy at which infection occurs are so closely related: nor that the formation of antibodies associated with the infection is similarly bound up. The development of recognisable agglutinins is of considerable importance in the control of brucellosis, and the observations recorded in the article are of much value. Evidence is given to show that infection may have been present for many months, even from the time of service, before agglutinins can be demonstrated in the later stages of pregnancy. From this finding it is somewhat disconcerting that so much reliance may be placed at times on the result of a single agglutination test. Thomsen's work gives us explanations of some of the disappointing findings in the control of brucellosis by the carrying out of agglutination tests and the isolation of non-reacting cattle: it also shows the great need for repeated testing over a period. It further helps us to understand why some animals, non-reactors before shipment, are found to react positively to the agglutination test on arrival in another country.

While these observations are valuable and interesting, much reliance must still continue to be placed on the agglutination test in giving an opinion on whether or not an animal is infected with *Br. abortus*. The agglutination test must still be regarded as the method of diagnosis, and importing countries will, doubtless, continue to attach considerable importance to the results of the test.

On the question of carrying out the tests, techniques and interpretations differ in different countries, and even in some parts of the same country. This sometimes results in expressions of opinion which vary among operators, especially concerning low or " border-line " titres. It has been felt for some years that agreed techniques, types of antigens and methods of interpretation between countries should be adopted. Thanks to the efforts of the Office International des Epizooties, attempts are now being made to come to definite arrangements on these points between different countries, and we look forward to the time, in the not distant future, when it will be possible to report that the necessary agreements have been obtained.

Complications in the significance of the results of agglutination testing arise in animals which have been vaccinated: these refer to the use of vaccines like that produced from *Br. abortus,* Strain 19, which gives rise to the formation af agglutinins. It is realised that such agglutinin production should follow the injection of the vaccine. A report given at the International Veterinary Congress last year showed the time taken for the reactions to disappear in vaccinated animals. Briefly, the findings were that: " Animals vaccinated as calves showed an incidence of about 25 per cent which retained titres of 1 in 20 or above for at least three pregnancies. The remainder showed negative reactions at titres below 1 in 20 by the time of their first pregnancy. About 25 per cent of animals vaccinated as heifers retained titres of 1 in 20 or above for at least four pregnancies. Many animals vaccinated as adults failed to show negative results or titres below 1 in 20 during the five years' observation."

Recently, there has been a change in the Canadian import regulations concerning agglutination results in cattle from this country. It is now stated, with regard to brucellosis, that: " Cattle up to thirty months of age will now be accepted into Canada from Great Britain, whether negative or positive to the blood agglutination test for brucellosis, provided that they have been inoculated with *Brucella abortus,* Strain 19, vaccine, between the ages of four and eight months, and also provided that a certificate to that effect is signed or endorsed by a veterinarian employed full-time by the Ministry of Agriculture and Fisheries. It is emphasised that this amendment applies to animals destined for Canada only."

GENERAL ARTICLES

EXPERIMENTAL STUDIES ON THE INCUBATION PERIOD OF INFECTIOUS ABORTION IN CATTLE

By AXEL THOMSEN, D.V.M.,

Chief of the Brucellosis Department, The State Veterinary Serum Laboratory.

In this paper the term " incubation period of infectious abortion in cattle " means the period from the infection of the fertilised animal (heifer) to parturition (abortion).

The statements in the literature concerning the length of this period vary greatly. The comment of Fröhner and Zwick in their " Handbook of Special Pathology and Therapy "—" Die Inkubationsfrist lässt sich nicht bestimmt obgrenzen "—illustrates the position.

The length of this period is given differently by various authors; furthermore, individual authors give very wide limits for its duration. Thus, McFadyean and Stockman (1909) state that it varies from 33 to 230 days (average 126 days), and Sv. Wall (1934) gives it as from 12 to 189 days. The cause of this variation is doubtless dependent upon various conditions, such as the weight of the infection, the virulence of the bacteria in the given case, the resistance of the animal, and probably the path of infection, e.g., oral, conjunctival, vaginal, cutaneous, or subcutaneous. The confusion regarding the variable incubation periods given in the literature is often due to the uncertainty of the exact time at which the infection occurred (e.g., Pröschold [1932]; Sv. Wall [1934]).

A factor which hitherto has not apparently been given much attention in this connection is the stage of pregnancy, i.e., the state of development of the fœtus, at the time of the infection (e.g., Beller and Zeller [1934]). This factor, as will be pointed out in this article, is of considerable significance. The present studies were prompted largely from the results of American experiments, as well as by the author's own work on the possibility of transmission of the infection through copulation, and also as a result of several experiments (Thomsen [1928]), which showed a considerable resistance of non-pregnant animals and animals in the first stage of pregnancy.

As early as 1936, the author was able to demonstrate (Thomsen [1936]) that the length of the incubation period was inversely proportional to the development of the pregnancy when the infection took place.

In order to confirm this observation and to investigate it thoroughly, further experiments have been carried out, and the total material now comprises 24 heifers, all of which were purchased from blood-tested, brucellosis-free herds. During the period of the experiment these animals were housed in the experimental station of the laboratory (" Tavlebakkegaard ").

In Figs. 1 to 5, the course of the infection in five aborting heifers is demonstrated: the heifers were infected at the time of service and fertilisation, the bull being infected, immediately before service, by injecting 10 c.c. of a

FIGS. 1-5.—Shows the course of the infection in five aborting heifers.

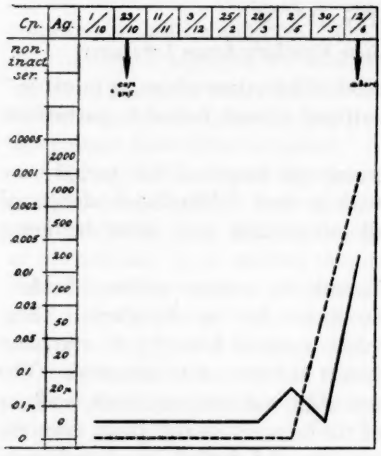

FIG. 1.—Heifer No. 288.
Copulation and infection on 22/10.
Abortion 11/6.
Incubation period: 232 days.

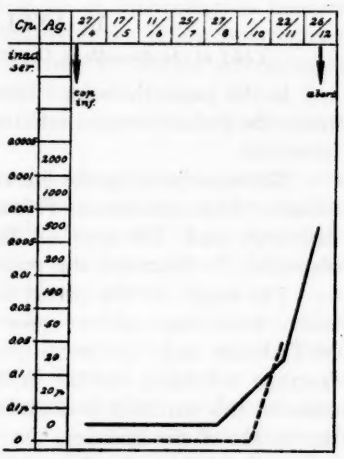

FIG. 2.—Heifer No. 159.
Copulation and infection on 19/4.
Abortion 26/12.
Incubation period: 251 days.

KEY TO ABBREVIATIONS

Heavy line is agglutination titres.
Broken line is complement fixation results.
Cop. = copulation (service).
Inf. = infection given.
Abort. = abortion.

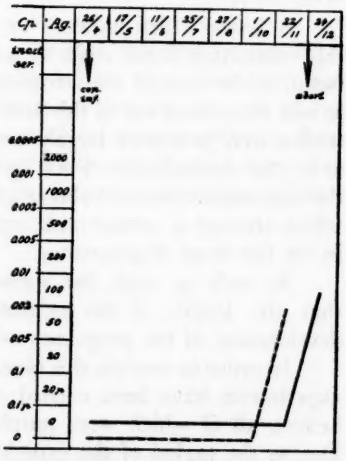

FIG. 3.—Heifer No. 58.
Copulation and infection on 25/4.
Abortion 20/12.
Incubation period: 239 days.

suspension of brucella-infected placenta into the prepuce: this is a massive infection when compared with natural infection during copulation.

All the five heifers referred to in the Figs. aborted, whereas five other heifers, infected in an exactly similar manner, calved normally: four of the latter animals failed to show any antibody formation in the blood. This finding indicates the resistance to infection at the time of service.

All the placenta from the five aborting heifers showed specific changes, and *Br. abortus* was demonstrated microscopically.

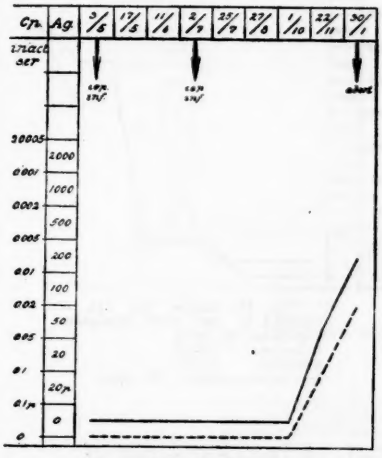

FIG. 4.—Heifer No. 193.
Copulation and infection on 3/5 and 2/7.
Abortion 29/1.
Incubation period. 211 days.

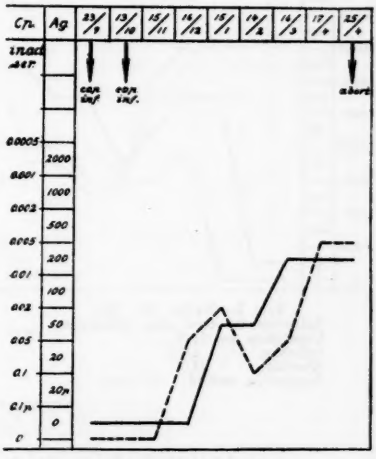

FIG. 5.—Heifer No. 33.
Copulation and infection on 22/9 and 13/10.
Abortion 24/4.
Incubation period: 193 days.

From the Figs. it will be seen that the incubation period following transmission at this time is surprisingly long, viz., from 193 to 251 days (average 225 days). This long incubation period explains why, in practice, abortions due to *Br. abortus* infection are seldom seen before the 5th or 6th month of pregnancy: this finding indicates the need for caution in considering the origin of the infection in new-infection in a herd.

A series of 19 heifers were infected orally and by way of the conjunctiva at different stages of pregnancy. Each heifer received by mouth 25 g. minced infected placenta suspended in saline: and, in addition, 2 or 3 drops of the same suspension were instilled into the conjunctival sac. For each infection fresh placental material, taken from two to four spontaneous abortions, was used.

While the routes of infection, the infecting dose, and, generally speaking, the infective material were similar for all 19 animals, the infection was carried out at different periods of pregnancy. This is shown in Figs. I to XIX, which also record the antibody production in relation to the time of the infections.

XUM

FIGS. I-XIX.—Record the antibody production in relation to the time of the infections.

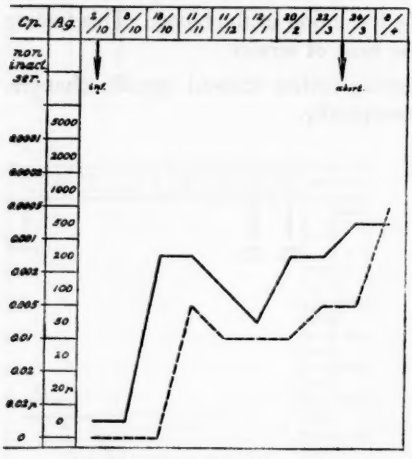

FIG. I.—Heifer No. 200.
Infection 10 days after copulation.
Copulation on 22/9.
Infection 2/10.
Abortion 23/3.
Incubation period: 172 days.

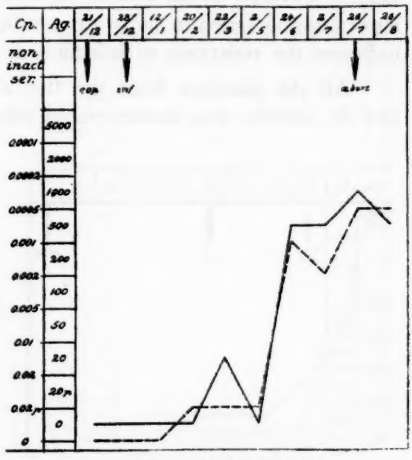

FIG. II.—Heifer No. 184.
Infected 21 days after copulation.
Copulation on 7/12.
Infection 28/12.
Abortion 25/7.
Incubation period: 209 days.

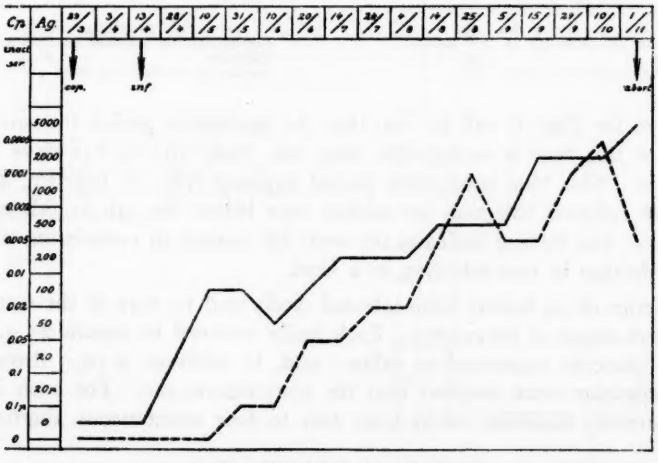

FIG. III.—Heifer No. 486.
Infection 21 days after copulation.
Copulation on 20/3.
Infection 10/4.
Abortion 1/11.
Incubation period: 205 days.

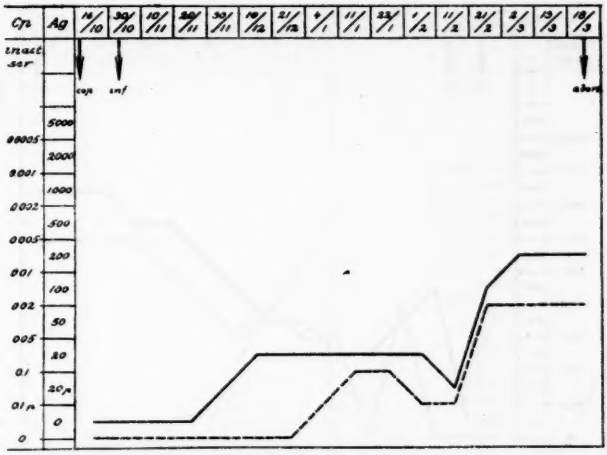

FIG. IV.—Heifer No. 482.
Infected 30 days after copulation.
Copulation on 27/9.
Infection 27/10.
Abortion 18/3.
Incubation period: 143 days.

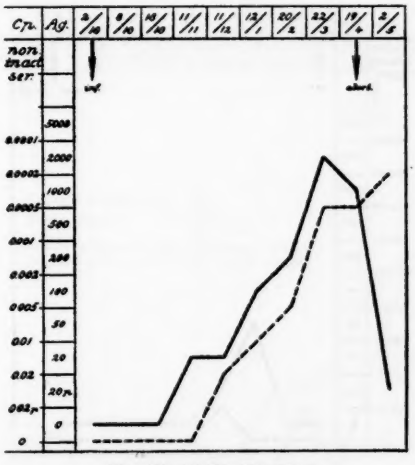

FIG. V.—Heifer No. 198.
Infected 34 days after copulation.
Copulation on 29/8.
Infection 2/10.
Abortion 19/4.
Incubation period: 199 days.

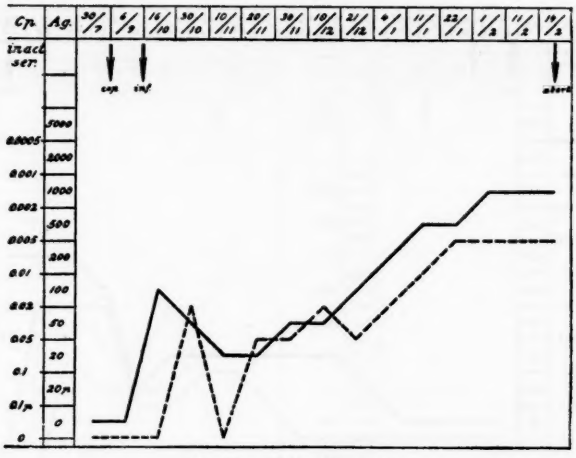

FIG. VI.—Heifer No. 475.
Infected 45 days after copulation.
Copulation on 14/8.
Infection 28/9.
Abortion 14/2.
Incubation period: 139 days.

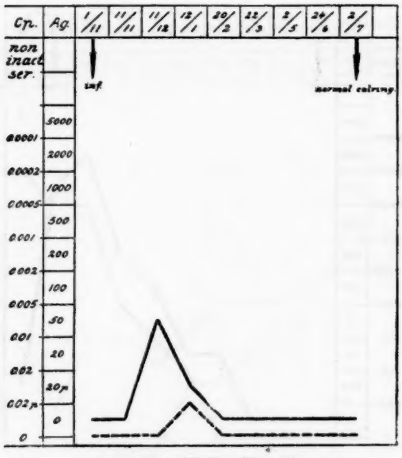

FIG. VII.—Heifer No. 196.
Infected 47 days after copulation.
Copulation on 15/9.
Infection 1/11.
Normal calving on 2/7 (290 days).
No infection of the uterus.

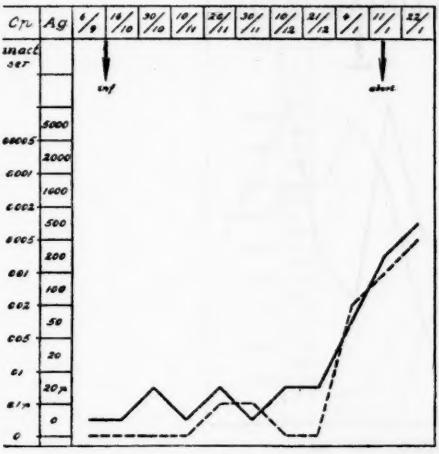

FIG. VIII.—Heifer No. 471.
Infection 61 days after copulation.
Copulation on 4/8.
Infection 4/10.
Abortion 10/1.
Incubation period: 98 days.

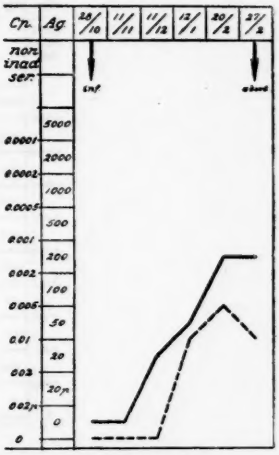

FIG. IX.—Heifer No. 197.
Infected 62 days after copulation.
Copulation on 27/8.
Infection 28/10.
Abortion 26/2.
Incubation period: 121 days.

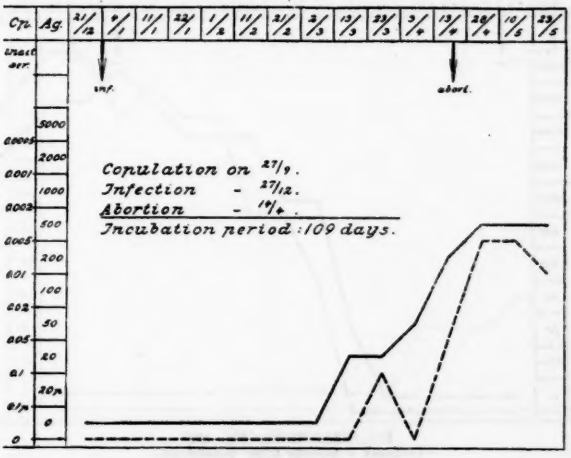

FIG. X.—Heifer No. 474.
Infected 91 days after copulation.

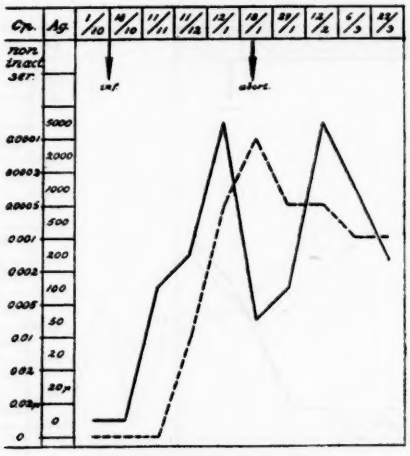

FIG. XI.—Heifer No. 284.
Infected 93 days after copulation.
Copulation on 7/7.
Infection 8/10.
Abortion 17/1.
Incubation period: 101 days.

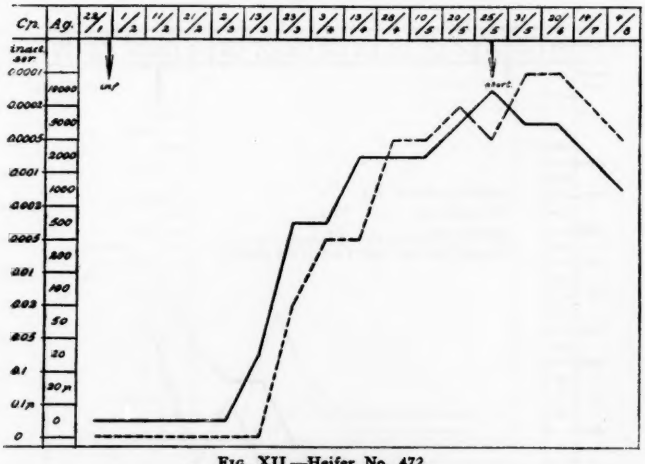

FIG. XII.—Heifer No. 472.
Infected 4 months after copulation.
Copulation on 28/9.
Infection 29/1.
Abortion 25/5.
Incubation periad: 117 days.

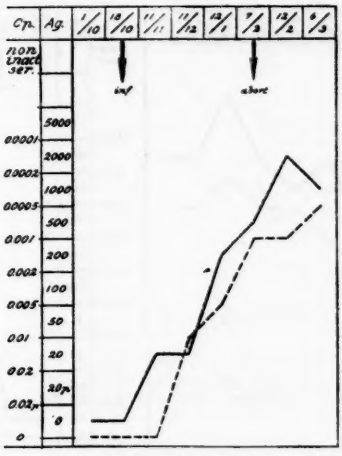

FIG. XIII.—Heifer No. 281.
Infected 4 months after copulation.
Copulation on 18/6.
Infection 18/10.
Abortion 7/2.
Incubation period: 112 days.

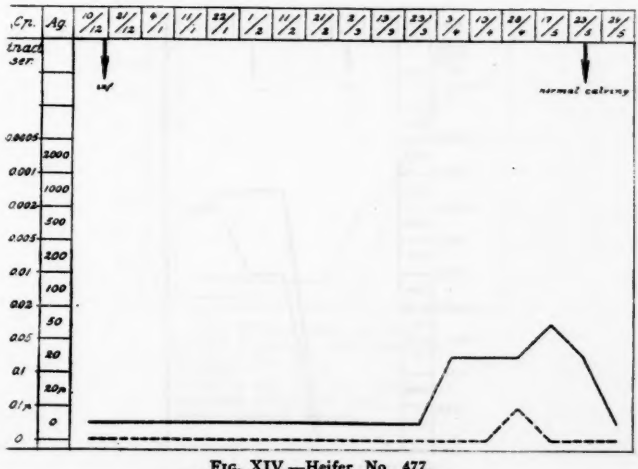

FIG. XIV.—Heifer No. 477.
Infected 5 months after copulation
Copulation on 14/7.
Infection 14/12.
Normal calving on 23/5 (314 days).
No infection of the uterus.

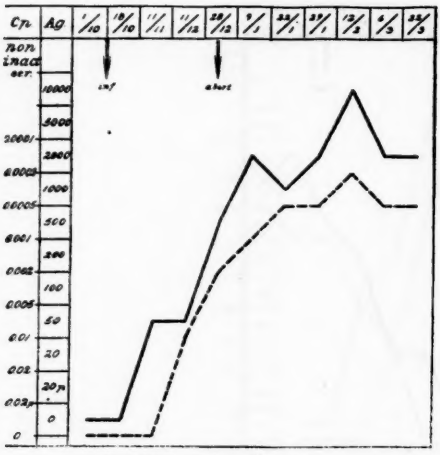

FIG. XV.—Heifer No. 287.
Infected about 5 months after copulation
Copulation on 2/5.
Infection 8/10.
Abortion 27/12.
Incubation period: 80 days.

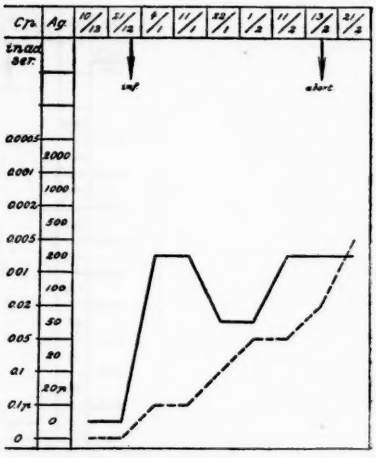

FIG. XVI.—Heifer No. 481.
Infected 6 months after copulation.
Copulation on 23/6.
Infection 24/12.
Abortion 13/2.
Incubation period: 51 days.

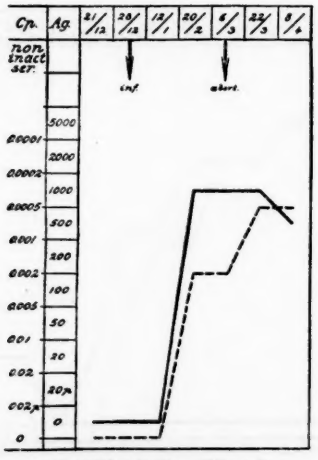

FIG. XVII.—Heifer No. 185.
Infected 6 months after copulation.
Copulation on 30/6.
Infection 28/12.
Abortion 5/3.
Incubation period: 67 days.

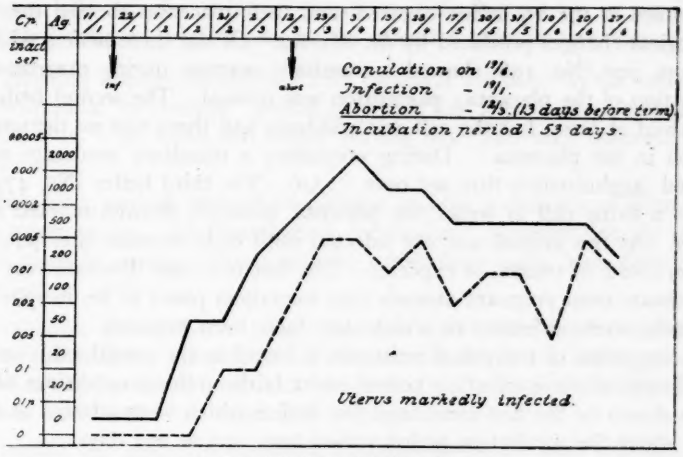

FIG. XVIII.—Heifer No. 480.
Infected 7 months after copulation.

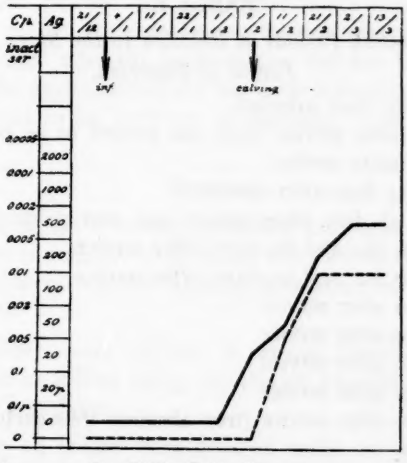

FIG. XIX.—Heifer No. 473.
Infected 8 months after copulation.
Copulation on 27/4.
Infection 27/12.
Calving on 9/2 (288 days, calf living).
Incubation period: 44 days.
Uterus markedly infected.

KEY TO ABBREVIATIONS

Heavy line is agglutination titres.
Broken line is complement fixation results.
Cop. = copulation (service).
Inf. = infection given.
Abort. = abortion.

Sixteen of the 19 heifers aborted, and their placentas showed the typical pathological changes produced by *Br. abortus*. Of the three heifers which did not abort, one (No. 196) showed a transitory reaction during pregnancy, but no infection of the placenta: parturition was normal. The second heifer (No. 477) calved at term, but the calf was still-born and there was no demonstrable infection in the placenta. During pregnancy a transitory moderate reaction appeared (agglutination titre not over 1 : 50). The third heifer (No. 473) gave birth to a living calf at term: the placenta, however, showed marked specific changes. As this animal was not infected until eight months after service, no abortion could, of course, be expected. The first two cases illustrate very clearly how resistant even pregnant animals may sometimes prove to be, in spite of the apparently severe infection to which they have been exposed.

A suggestion of individual resistance is found in the considerable variation in the length of the incubation period under fairly uniform conditions of infection, as shown by the first-mentioned five heifers which were infected at service, and in which the incubation period varied from 193 to 251 days.

Table I shows definitely that the length of the incubation period is inversely proportional to the development of the fœtus at the time of infection.

TABLE I.

Average Incubation Periods in Relation to the Stage of Pregnancy.

Heifers.	*Period of Infection.*	*Days.*
5	At service (bull infected)	225
2	10 days after service (only one proved to be pregnant)	172
2	21 days after service	207
2	30 and 34 days after service	171
2	45 and 47 days after service (one was resistant) ...	139
2	2 months (61 and 62 days) after service	110
2	3 months (91 and 93 days) after service	105
2	4 months after service	115
2	5 months after service	80
2	6 months after service	59
1	7 months after service	53
1	8 months after service (true abortion thus excluded) ...	44

Some rather interesting information is obtained from a study of the serological reactions, especially their appearance in relation to the time of the infection.

In particular, it is noteworthy that the interval between inoculation and the occurrence of the reactions has often been long: in some animals it was several months, even over half a year, before any evidence of infection was seen in the serological picture. The period between the demonstration of the reaction and the occurrence of the abortion is sometimes very short. As is well known, in some cases reactions do not appear until some time *after* the abortion has taken place (Thomsen [1928]). The very long intervals without reactions are

seen especially in the relatively moderate infection set up at service with artificially "infected" bulls, i.e., in non-pregnant animals (see Figs. 1-5).

Following massive inoculation into pregnant animals, on the other hand, the "serological incubation" period as a rule has been a few weeks (2 to 6) only (see Figs. I to XIX), but longer periods have also been observed (heifers 184 and 474).

These long periods between inoculation and the occurrence of demonstrable blood reactions, i.e., the "serological incubation," illustrate clearly the very limited value of serological tests and the misleading results from them in cases of infection at service.

It follows, therefore, that only very slight significance can be attached to a single negative blood reaction in an animal, the origin and other information of which are unknown or uncertain: *protracted clinically and serologically latent infections have both to be taken into account.*

Usually, the antibody formation increases gradually until abortion occurs, after which the increase often continues for a short time before the titre begins to fall.

The complement fixation and the agglutination curves are usually fairly concurrent, but, as a rule, the complement fixation reaction cannot be demonstrated until some time after the agglutination test has become positive. For this reason alone, the agglutination test has to be looked upon as the more suitable of the two reactions for purposes of diagnosis: it is, indeed, more widely employed.

Summary

The incubation period of bovine brucellosis and the corresponding antibody formation, especially in relation to the exact time of infection, are demonstrated by experiments and blood tests on 24 sexually-mature heifers, originating from brucellosis-free herds.

Five of the heifers were infected at service, infectious material (minced placenta, suspended in saline) being introduced into the prepuce of the bull immediately before copulation.

The remaining 19 heifers were infected orally, as well as by way of the conjunctiva, at different periods of pregnancy.

A table is given which shows quite definitely that the length of the incubation period is inversely proportional to the stage of development of the fœtus at the time of infection: *the younger the fœtus at the time of infection, the longer is the incubation period.* This is shown by the finding that the average incubation period was 225 days in the animals that were infected at service, and only 53 days in a heifer that was not infected until seven months after service.

The longest incubation period recorded in the present observations is 251 days. The long incubation period, especially following vaginal inoculation at

service, explains why, in practice, few brucella abortions are found before the 5th or 6th month of pregnancy: this makes it difficult to show the source of infection in a new-infection herd. A period of months, even half a year or more ,sometimes elapses before antibody formation becomes evident following infection: this applies especially to moderate infections at service.

In some cases antibodies cannot be demonstrated until abortion has taken place. This fact has been known for a long time, but probably it has not been realised that in such cases the infection may have persisted for many months, perhaps even from the time of service, as is suggested by the present findings.

On the basis of the present experiments it seems justifiable to claim that the value of serological tests is very limited, and the result may even be misleading when a single examination is carried out, especially in animals infected at service or those with an obscure history.

The curves of the agglutination and complement fixation reactions are fairly similar, and show no particular difference in the value of either test. As a rule, however, complement fixing substances cannot be demonstrated until some time after the agglutinins have appeared in the serum. For this reason, as well as for its simplicity, the agglutination test is preferable for routine diagnostic purposes.

REFERENCES

McFadyean, J., and Stockman, S. (1909): *Rpt. Dept. Ctle. Epizootic Abortion, London, 1,* 14.
Wall, S. V. (1934): *Skandinavisk Veterinärtskrift, 24,* 453.
Pröschold (1932): *Berl. Tierärzt. Wsch.,* 573.
Beller and Zeller (1934): *Münch. Tierärzt. Wsch.,* 1.
Thomsen, Axel (1928): *Den Kgh. Veter. og Landbohjskoles Aarsskrift.,* 76.
Thomsen, Axel (1936): *Maanedsskrift for Dyrlaeger, 48,* 225.

THE PUBLIC HEALTH ASPECTS OF POULTRY DISEASE

BY W. P. BLOUNT, PH.D., F.R.C.V.S., F.R.S.E.

I.—Introduction

MOST persons attending this Congress will appreciate the difference in procedure and practice between routine meat inspection and that for poultry. They will know that whereas 98 per cent of red meat undergoes rigid and efficient inspection, most poultry escape any regular inspection at all, except in certain cities. They will be aware, of course, that it is perfectly legal for any butcher, fishmonger or poulterer to offer for sale game or poultry without any inspection; indeed, less than 5 per cent of all birds eaten to-day will have seen a meat inspector's knife, in the country as a whole.

It is an offence for a householder to kill and eat any pigs he may possess, and worse still, perhaps, is the case of the genuine beef farmer should he attempt to utilise for his household any steer which falls by the wayside; in

the case of the domestic poultry keeper, no rule, legislation or law of any sort is prescribed which makes the killing or eating of poultry an illegal act. He or she may pick and choose the bird to be slaughtered at leisure and no one will worry whether it is young or old, ailing or healthy—provided the act of killing is performed expeditiously, mercifully and efficiently.

From this preamble it will be realised that there is no legal requirement for dead poultry necessarily to be examined prior to sale in shops or elsewhere. On the other hand, if a carcase is exposed and it appears reasonable for a meat inspector to wish to examine such a bird he is entitled to do so, and, furthermore, it is presumably an offence for anyone to expose for sale any carcase which is known to be diseased. In the City of London, for example, meat inspectors regularly "vet" poultry at Smithfield and Leadenhall markets, etc., and not infrequently they make an incision to view the abdominal organs of any poultry if suspicion exists of the presence of tuberculosis, etc. Similar examinations may be made at any wholesale or retail establishment and occasionally the proprietor of such premises is asked to withhold the sale of a given consignment until after it has been officially examined.

But what of the several diseases known to afflict both poultry and man—the many maladies which may be transmissible from avian to human hosts? Tuberculosis, for example, or salmonellosis, erysipelas, favus, aspergillosis, thrush or psittacosis? What about cholera, typhoid, helminth infestations, malaria, and also the many tumours, including the cancer (carcinomata) group? Is there any reasonable chance that the consumption of a jaundiced chicken or one which had died from rickets, nephritis or paralysis would reproduce a similar complaint in man; or that a pullet suffering from egg peritonitis would render anyone eating it liable to suffer also from peritonitis? Since one cannot necessarily assume that a bird will go into the oven to be roasted before being eaten, or even carefully boiled to the delectable standards of "Mrs. Beaton" or "Good Housekeeping," is there any real danger from eating par-boiled carcases, or even more so from handling poultry affected by any of the above-mentioned diseases? Finally, and from a somewhat different angle, is there likely to be any danger from eating meat which has been under the influence of either female sex hormones or iodinated casein-containing preparations?

The position of the eating egg must be considered of equal importance with that of table poultry, perhaps more so, in view of the fact that there is an official ration of eggs (or an egg) to all members of a household. There is also the special risk of infection peculiar to imported dried or frozen eggs and table poultry.

Perhaps it would form too much of an anti-climax to suggest at this stage of the paper that one should not get unduly alarmed at the thought of contracting one or more of the diseases mentioned earlier. Yet the fact remains that the number of cases of disease in man directly attributable to either handling or eating poultry meat or eggs is small, and then limited generally

to certain bacterial infections which usually remain as isolated cases and do not flare up as an epidemic. Certainly this is true for all practical purposes in every case except where *Salmonella* infections of egg origin are concerned.

Perhaps this comparative immunity on our part is due to rationing rather than resistance, although one assumes rightly or wrongly that micro-organisms parasitic to birds seldom find as favourable a breeding-ground when transferred to human tissues. Let us, however, face the facts squarely—from what diseases do poultry die to-day?. At the B.O.C.M. hen battery laying trials, where 1,000 pullets, from 150 or more farmers scattered throughout Great Britain, are housed each year, the mortality has been about 12 per cent, and the chief post-mortem findings compared with those of the 10.8 per cent mortality at the 1947/48 National Laying Test were as follows:—

	B.O.C.M. Hen Battery Laying Trials.		National Laying Test.
	1946/47.	1947/48.	1947/48.
Diseases of the reproductive system, including egg peritonitis, internal laying, impacted oviduct, prolapse, etc. ...	28%	17%	32%
Fowl paralysis group, including lymphomatosis, leukæmia and splenomegaly ...	13%	27%	16%
Other tumours (including carcinomata) ...	13%	12%	5%
Degenerations of the liver, including internal hæmorrhage	12%	6%	3%
Diseases of the excretory system, nephritis and visceral gout	6%	12%	9%
Diseases of the digestive system, including vent gleet	6%	8%	6%
Diseases of the circulatory system (heart failure, etc.)	—	—	4%
Tuberculosis	3%	—	2%
Diseases of the respiratory system	—	—	2%
Miscellaneous	19%	18%	15%

II.—Bacteriological Infections

Tuberculosis.—The causal organism of tuberculosis in poultry differs markedly from that of bovine animals because it never becomes generalised when introduced into cattle, it never invades the udder (and therefore the milk), and it rarely infects either the herdsman or any other human being. At the same time there are, throughout the world, records (sometimes of doubtful authenticity) of tuberculosis in man due to *Mycobacterium avium*, but for every 100,000 cases due to human or bovine strains of the tubercle bacillus there is probably only one of avian origin. Yet, curiously enough, avian

tuberculosis does spread comparatively easily amongst certain mammals, e.g., pigs. Pullin, in Canada, for example, found 70 per cent of 232 lymph glands from pigs tuberculous, and of these 104 contained sufficient organisms of the avian type to produce tuberculosis in chickens And there are, of course, a number of records of avian infection in pigs in this country. Curiously enough, following inoculation with lesions from pigs, the thymus gland may show more advanced changes than any other avian tissue.

One cannot, from the meat inspector's standpoint, dismiss the disease too lightly, particularly when emaciation of the affected poultry carcase is not necessarily present. Indeed, some 10 to 15 per cent of tuberculous poultry die in good or even fat condition, and their sudden death is often due to internal hæmorrhage from a ruptured, fatty, tuberculous liver. It will be remembered that the incidence of tuberculosis in poultry is greater in the higher age groups, particularly in females, so much so that it may reach 15 per cent in hens five years old or over.

It is fortunate, perhaps, that modern poultry practices are tending to eliminate tuberculosis from poultry. This is because many general farmers are wisely keeping all-pullet flocks, in contrast with other farm flocks, where a goodly proportion of the so-called laying stock consists of older hens, often seen hanging around the granary or cart-shed.

The gradual disappearance of the semi-intensive method of poultry husbandry—whereby small areas of ground (adjacent to poultry houses), thickly populated with adult poultry year after year, become increasingly "sour" and disease-organism contaminated—is to be welcomed. Hen batteries and intensive or portable housing systems are also favourable in the battle against avian tuberculosis. It is fortunate, too, that, unlike the human race, when poultry become tuberculous they generally lose their sexual instincts and egg production ceases. (Chicks have been born from tuberculous eggs, but they have always died within a few days of hatching.) Tuberculosis, therefore, is not often encountered in "table" poultry (both because of their youth and intensive housing conditions), and although it is found in older hens, the danger to man is minimised greatly, since the carcase is cooked before being eaten. Furthermore, as human beings are extremely resistant to infection by the avian tubercle bacillus, little trouble need be expected.

Salmonella Infections.—As a genus of pathological significance, this is certainly the largest involving poultry and, although there is an odd record or two of *S. pullorum* infection involving man, generally speaking one neglects any pathogenic relationship between bacillary white diarrhœa and the human race. Similarly, with *S. gallinarum,* causal organism of fowl typhoid, but the position is less happy where some other salmonella are concerned.

In *S. enteritidis-Gærtner* we have an occasional invader of poultry products and a possible agent for human infection primarily of intestinal origin, but as it is also the active biological component of a well-known proprietary rat killer,

the poultry angle is clearly of importance. Both E. O. Jordan (1925) and R. S. Spray (1926) consider that several human epidemics have been traceable to the use of commercial rat "viruses." But this was doubtless at a time when the handling of such potentially infective material was not necessarily as good as to-day.

In *S. thompson,* nowadays considered to be a serious menace to chick rearing in many areas of the country, we have a germ whose primary host is man himself, and it is therefore of interest to speculate whether poultry infect man, or vice versa. We do know, however, that it is not an ovarian infection in the hen, as so often occurs in bacillary white diarrhœa (Pullorum) infections, but that contamination of eggs usually arises secondary to intestinal infection. Whereas at one time the main site of actual infection was considered to be the cloaca, it is probably more correct to visualise most infection occurring via dirty (contaminated) nests at a time when the freshly-laid eggs are both warm and moist. Since the malady in poultry, other than chicks, is not characterised by a blood stream infection, once a carcase has been "drawn" it is comparatively clean, and certainly free from *S. thompson* infection once it is cooked.

Paratyphoid infection, due to *S. typhi-murium,* can be serious in all farm poultry, particularly in chicks and ducklings, and, as is well known, contaminated eggs laid by adult ducks occasionally account for death in man. As recently as 1946, Crowe describes an outbreak of *S. typhi-murium* food poisoning involving twenty-three persons, and the evidence suggested that the source of infection was a hen's egg which had been incorporated in a butter substitute. Unfortunately, blood agglutination tests to detect adult carrier ducks, etc., are rather unsatisfactory, and the use of sulfa drugs, although frequently spectacularly succesful in the early rearing stages, is often of little or no value in cleaning up adult infection of the ovary. *S. enteritidis dublin* has recently been the cause of rearing losses in chicks, but cattle appear to be the usual reservoir of such infection, and it is not common in adult poultry.

Many other species of *Salmonella* have been identified, particularly in imported dried egg products, but I leave their description and place as ætiological agents in the consideration of human infection to others better qualified to discourse on the subject.

Curiously enough, *S. thompson* infection in poultry in the U.S.A. is virtually unknown.

On the whole, one must view salmonella infections of the paratyphoid type in chickens, ducks, turkeys and geese with considerable suspicion where spread to man is concerned, but from a meat inspector's angle it is more often eggs or egg products which require vetting, and not the animal carcases as such.

Erysipelas in poultry is rare, except in turkeys, and recently several fresh cases have been reported in the U.S.A. and Canada, as well as the occurrence of suspicious cases in this country.

Erysipelothrix rhusiopathiæ, the causal agent, is common enough in pigs, and it has been identified also as the cause of polyarthritis in sheep. It has been found on the slime of both fresh and salt water fish, and even in fish-meals, and, of course, laboratory workers have become infected by it occasionally during the course of their work. But erysipelas infection in turkeys is rather a new feature, and, therefore, a fresh source of trouble to poultry farmers; one wonders, therefore, whether the handling of infected carcases, under cold storage conditions, may be dangerous. As regards treatment, some turkeys have responded well to penicillin therapy—20,000 units twice daily. The surprisingly high incidence (90 per cent) in male turkeys is not due to any special susceptibility, but merely an indication of the ease with which aggressiveness in males leads to local injuries, thus giving the organism easy access to the tissues generally.

III.—Less Important Infections

Botulism.—Birds act as passive carriers of infection, for the organism is present in some healthy intestines. T. G. Hull (1930) states: "In twenty-two instances, the outbreaks in chickens were caused by careless dispositioning of food, which had caused human outbreaks, and to which the chickens had access." Boiling kills the toxins, and the organism as such is harmless to man.

B. morgani is an organism allied to *B. coli,* and appears to be associated with the cause of some epidemics of diarrhœa in children, from whose fæces it can be isolated. It has been recovered in this country in pure culture from both chicks and adult birds by McGaughey, a veterinary professor now resident in Ceylon. But there appear to be few records of its spread to man, even though its bacillary white diarrhœa-like characteristics are such as to render recovery of the organism from the ovary possible in at least one instance.

Fowl cholera, due to *Pasteurella* infection, is responsible for acute outbreaks of disease in poultry, and it may also be the cause of chronic localised infections of the wattles, nares and joints. But man is not generally considered to be susceptible to infection by *P. avicida,* although there are claims for the organisms to have been isolated from local cutaneous abscesses.

Listerellosis in chickens is due to *Erysipelothrix* (*Listeria*) *monocytogenes,* an organism which has also been isolated from rodents, rabbits, pigs, cattle, sheep and man. Culturally and morphologically, this organism is very similar to *Erysipelothrix rhusiopathiæ,* except that the latter is non-motile. In man it has been associated with fatal cases of *Meningo-encephalitis, Otitis media,* etc., but it is probably not the organism responsible for infective mononucleosis. Paterson recorded the existence of *Listerellosis* in four flocks in England in 1937, in which the affected birds were showing liver degenerations, œdema and emaciation.

Pasteurella pseudo-tuberculosis is a rare cause of infection in poultry, and also man. It was first isolated from the latter in 1833 from the forearm of a

child, but its occurrence in many birds is well known, e.g., finches, pigeons, pheasants, swans, etc., and even in a blackbird.

Pseudomonas pyocyaneus has been isolated from epidemics involving chicks and adult birds, but it is normally considered of low virulence and readily isolated from polluted water, where it may be saprophytic.

Staphylococcus aureus, an organism recoverable from some joint lesions in pheasants, ducks, geese and poultry is also well known for its association with abscesses, etc., in man. But the incidence of its spread from poultry to man is negligible. On the other hand, there appear to be records of hatcherymen suffering from impetigo, abscesses or boils, having infected newly-hatched chicks to cause them to suffer from omphalitis.

IV.—Virus Infections

Psittacosis occurs in many avian species, including parakeets and other caged varieties, pigeons, and even domestic hens; and although the numbers of affected pigeons, for example, is at times demonstrably high, actual transfer of the disease to man is far less common in this country than one would anticipate. Indeed, even pigeon fanciers with " positive " stock seldom suffer from psittacosis, and it is also quite a rare disease in hens. Perhaps this is allied to the story of undulant fever, in which there is a large reservoir of *Brucella* infection in domestic cattle and unpasteurised dairy products, yet the number of farmers who actually suffer from undulant fever is low. Furthermore, even the practising veterinarian dealing with obvious cases of metritis and retention of the after-birth known to be of *Brucella* origin, seldom suffers patently, although his blood titre may be conspicuously positive. It is probable that repeated exposure to the causal organisms—no matter whether they are of virus or bacterial origin—gives some degree of immunity against forthright infections, particularly since sub-clinical infections may pass unnoticed. Nevertheless, when cases of psittacosis do occur in man, his acquisition of the infection from avian species is doubtless one of the first thoughts of his medical practitioner. The majority of psittacosis infections occur in middle-aged people, children under ten in this country having a low susceptibility: the disease is also generally milder in children than in adults.

As a result of the 1929/30 improvements in import and quarantine regulations of many countries, much good was done to curtail the infection previously contracted from members of the parrot family. Since that time, according to Meyer, eight new fatal psittacosis cases of parakeet origin have been recorded in this country between the years 1931-46, whereas there were over four hundred similar cases in Germany, related mainly to the exhibition and sale of locally bred parakeets. Meyer's excellent account of the disease can be read in Biester and Schwarte's " Diseases of Poultry."

Newcastle Disease (*Pneumo-encephalitis*).—America has had more outbreaks of Newcastle disease than any other country, and one would have

thought that if there was any real danger to man from the virus concerned, numerous cases would have been reported. Yet for some years the only record of infection has been one in which a laboratory worker suffered from conjunctivitis following a splash of virus-containing material into the eyes. Furthermore, laboratory workers carrying out routine H.I. tests often handle the live virus, with no special care, and so far they have remained quite free from obvious infection. One can assume, therefore, that there is no likelihood of real danger when handling Newcastle disease infected carcases.

Curiously enough, there is a slight biological relationship between poliomyelitis of man and Newcastle disease of poultry, in that serum from the former disease tends to neutralise the virus of the latter. But, by no stretch of imagination can one relate outbreaks of infantile paralysis with the present high incidence of Newcastle disease, in either this country or the States.

Neoplasms.—Tumours of a neoplastic nature are very commonly present in poultry at post-mortem examination; indeed, they represent to-day approximately one-third of the whole pathological picture in laying stock. The *Avian Leucosis Complex* embracing all forms of fowl paralysis and so-called "lymphatic leukæmia"—better known to the poultryman as big liver disease—forms the major pathological picture, but there is an increasing incidence of neoplasms crudely classed as "cancer." Carcinomatosis of the ovary, mesentery, pancreas, liver and lungs are prominent, but it is fortunate that none of these tumours is transmisible to human beings. Large numbers of poultry suffer from fowl paralysis in its typical paralytic form (neurolymphomatsis-gallinarium), and apart from a conspicuous enlargement of certain major nerves or plexuses, the carcase is normal and perfectly fit for human consumption. I have, indeed, eaten many such birds willingly, and I hope to repeat this pleasure frequently!

Whether there is any danger in handling birds which have died from true leukæmia is still a matter of conjecture, because although one would not assume this possibility, because of the great genetic gap between the two species concerned, it is apparently true that Thiersh has reproduced erythomyelosis in hens following the inoculation of blood from a chronic case of human myeloid leukæmia. And after three such passages fowl paralysis then appeared as a complicating feature!

V.—Fungus Diseases

Aspergillosis.—Although there are upwards of four hundred different species of *Aspergillus,* most of them are pure saprophytes and quite non-pathogenic. Indeed, some are used commercially as agents of fermentation, and one, *A. oryzæ,* is used in the preparation of a Taka-diastase ferment.

About sixty parasitic members of the genus may be pathogenic, and amongst birds the pigeon appears to be one of the most susceptible species, and the spread of aspergillosis to pigeon fanciers is well known. This, however,

was more common fifty years ago amongst breeders who fed pigeons from their own mouths with seeds. Inhalation of aspergillus spores, however, could occur with the improper handling of any infected carcase, particularly if any pre-existing disease of the person's respiratory system was present.

A. fumigatus is generally considered to be the most pathogenic species, and it is found abundantly in soil, and agricultural products generally. It alone, among the many aspergillus species believed to be pathogenic, is the one which has fulfilled Koch's laws. Aspergillosis in adult poultry is uncommon, but it accounts for quite a number of outbreaks of nodular "brooder" pneumonia among chicks. In Canada and the U.S.A., aspergillosis of turkeys is common, but it is a rare disease of turkeys in this country.

Favus.—Favus is normally considered communicable from poultry to man, but it is seldom seen in the former species to-day, owing to good housing and sanitation. When cases do occur they only appear to involve isolated birds whose white combs and faces are distinctive enough to attract even the poultryman's attention, after which treatment or slaughter are usually initiated promptly. In his book, entitled "Fungus Diseases," H. J. Jacobson states, however, that that variant member of the genus *Achorion* responsible for favus in poultry, *A. gallinæ,* "has never been known to cause diesase in man. The common cause of human favus is *A. schonleini,* affecting the scalp, body and nails. Presumably, therefore, unless the invading fungus in avian favus is either *A. schonleini, A. quinckbanum* or *A. gypseum*—all known to produce lesions in man—there is no danger in handling poultry affected by "White Comb." On culture, *A. gallinæ* grows as a white, velvety disc, generating a reddish pigment, which distinguishes it clearly from other allied fungi.

Sporotrichosis.—Often characterised in man by cutaneous or subcutaneous granulomatous lesions, sporotrichosis is an infectious disease due to one of several fungi of the genus *Sporotrichium.* First recognised well over a hundred years ago, sporotrichosis is more common as an occupational disease in gardeners, farmers and stablemen, etc. Healthy persons generally resist infection, which is often introduced as the result of a wound or injury.

Sporotrichiæ are pathogenic to many animals, including fowls and pigeons, and in one instance the bite of a parrot was the cause of infection. So far as the writer is aware, no case has been recorded in poultry in this country.

Thrush.—In man, thrush is characterised by oral lesions of *Monilia* origin, and it is usually an acute condition. Lesions frequently resemble false membranes, but generally respond well to local treatment. Thirty species of *Monilia,* varying greatly in their pathogenicity and virulence, are known, and they are found usually on dead leaves, fruit, decomposing wood, excreta, etc., and their relationship to human disease goes back as far as 1839, when the parasite was known as *Oidium albicans.* It is, however, a common saprophyte, but may be associated occasionally with thrush in chickens, when the disease causes

diphtheritic lesions in the crop. Spread of the disease to man from this part is most unlikely, since the crop is usually discarded at eviscerating time.

VI.—Non-Specific Maladies

What is the danger, if any, to man in handling birds which die from the many non-specific ailments and infections which account for so large a proportion of deaths in commercial flocks?. One thinks immediately of such diseases as egg peritonitis, impaction of the oviduct, nephritis, pneumonia, enteritis, etc. And what about the chances of "poisoning" oneself by eating such birds after cooking?

During the last war, when red meat was in short supply, and poultry meat even more scarce, many people were faced with occasional poultry losses, sometimes due to mismanagement, e.g., impactions of the crop and gizzard, fractures of the leg, "internal-layers," prolapse, etc. At that time, as is the case to-day, all meat was very precious, and many of these birds therefore were killed, cooked and eaten. No harm resulted, but one soon learned that a "sick bird"—no matter from what cause—rarely, if ever, cooked tenderly. Most of the birds were tough, in spite of their youth, and it was noted that even a bird with a broken leg failed to reveal any attractive flavour or tenderness if allowed to live many hours before being killed for the table.

VII.—Rejects

A meat inspector will naturally reject all emaciated and thin carcases, and he presumably eliminates any birds which have *died* from disease in contrast to those normally *killed* for table purposes. Also any characterised externally by the obvious presence of pus, gangrene, ulceration, abscesses, jaundice, tumours, "white comb," bruising with frank hæmorrhage, fractures and dropsy. But the rationale for such a procedure is sometimes more psychological than sanitary, for the actual or even the potential danger to man is often slight. What his action will be on meeting cases of osteopetrosis, bumblefoot, gout proper, one-eyed roup, fowl-pox or advanced scaly leg, is a matter of conjecture, bias or experience.

From the internal view of the carcase gained as a result of incising the abdomen, the inspector will condemn also all poultry showing dropsy, peritonitis, pustular involvement of the air sacs, aspergillosis, visceral lymphomatosis, neoplasms, visceral gout, tuberculosis, multiple bowel tumours, and acute internal hæmorrhage. But what about the hen showing only one of the following conditions: a bacillary white diarrhœa ovary, patchy pneumonia or congestion of the lungs, lymphomatosis of the lungs, splenomegaly, nephritis, cystic Bursa of Fabricus, localised enteritis, fatty degeneration of the liver, proventriculitis (cystic), impaction of the crop, gizzard, ureter or oviduct? And suppose the abdomen appears normal except for a shell-less egg or two? Perhaps one easy way out of this problem is to pass only carcases which show

normal features—realising, of course, that even this more drastic rule would not be foolproof. (The veterinarian may encounter from 5 to 15 per cent of poultry post-mortem examinations in which even after a detailed inspection of all organs he still fails to find any obvious cause of death.)

Omitting altogether the question of diseased poultry, there is the public health angle to be considered, where fattening by female or other sex hormones is concerned. The use of stilbœstrol implantations into the upper portion of the neck is common enough in the U.S.A., but it has been temporarily banned in Canada because of the danger of using preparations which might be carcinogenic in action. In this country a number of veterinary surgeons and poultry farmers have tried stilbœstrol for fattening table poultry, and the result has been successful on very many occasions. At our own demonstration farm at Stoke Mandeville, for example, we have had fourteen-week-old cockerels weighing $5\frac{1}{4}$ lb.—half a pound heavier than the controls—after incorporating œstradiol preparations in the feed in milligramme doses daily for six weeks prior to killing. It is felt by the writer that the medical profession should make a public statement as to those female hormones which are safe and those which are not suitable for the above purpose.

VIII.—Summary

Summarising the whole position of the public health aspect of poultry diseases, one can view this problem from both (*a*) the danger to the public of the consumption of poultry meat as normally cooked; and also (*b*) the danger from handling infective carcases either by the meat inspector himself or by the housewife prior to cooking the bird concerned.

In either case one is excluding, of course, the possibility of acquiring infection of avian origin from eggs—fresh, frozen, dried or otherwise processed. This latter angle concerns intimately the salmonellosis problem, which is of very considerable importance and one which I have not attempted to discuss.

In my view, there would be little danger to the public from the consumption of uninspected, cooked poultry meat as normally practised in this country. There is some slight danger to persons handling carcases, which no doubt would be increased if the person concerned had open wounds, however minor, of the hands. This would apply particularly to the production of erysipeloid lesions of *E. rhusiopathiæ* origin. The threat to the public from the consumption of tuberculous poultry is probably more imaginary than real, because of man's very considerable natural resistance to this form of tuberculous infection. The dangers of psittacosis are negligible when one is dealing with poultry—in contrast with the handling of infected parrots, or possibly, on occasions, pigeons. In view of Jacobson's statement concerning the genus *Achorion* relative to favus, there is much less danger than hitherto supposed from handling cases of "white comb," and the chance of thrush or sporotrichosis spreading to man from birds is indeed rare. On the other hand, if a person carelessly sniffed at a carcase infected by aspergillosis, inhalation of infective spores could take place.

On the whole, therefore, the danger to man from diseases of avian origin is slight; although doubtless the law allows a qualified meat inspector to condemn many carcases which would be quite incapable of inducing ill-health were they to be cooked and eaten.

REFERENCES

Bankier, J. C. (1946) : *Canad. J. Comp. Med., 10,* 164.
Blount, W. P. (1937) : "Diseases of Poultry." Baillière, Tindall & Cox, London, 21s.
Buxton, A. (1938) : "Avian Salmonellosis." Proceedings VIIIth. World's Poultry Congress. Vol. 1,609.
Crowe, M. (1946) : *J. Hyg. Comb., 44,* 342.
Field, H. I. (1949) : *Vet. Record, 61,* 109.
Graham, Levine and Hester (1939) : *J.A.V.M.A., 95,* 211.
Hagan, W. A. : "The Infectious Diseases of Domestic Animals." Comstock Publishing Co., Ithaca, N.Y., 1945. 33s.
Hull, T. G. : "Diseases Transmitted from Avians to Man." Charles C. Thomas, Baltimore, Maryland, 1930. 21s.
Jacobson, H. P. : "Fungus Diseases." Charles C. Thomas, Baltimore, Maryland, 1942. 25s.
Jordan, E. O. (1925) : *J. Inf. Diseases, 36,* 309.
Martin, C. R. A. : "Practical Food Inspection," Vol. II. 3rd edition. H. K. Lewis, London, 1948. 18s.
Paterson, J. S. (1937) : *Vet. Record, 49.* 49.
Pullin, J. W. (1946) : *Canad. J. Comp. Med., 10,* 159.
Spray, R. S. (1926) : *J.A. Med. Ass., 86,* 109.

Discussion

Dr. R. F. Gordon (Poultry Research Station) who opened the discussion, said it was always a pleasure to listen to a paper by Dr. Blount on any aspect of poultry husbandry; Dr. Blount had not only a wide knowledge of the poultry industry, but he had, in particular, a wide grasp of its commercial implications, and, in addition, he had shown that he was a facile and lucid speaker, with his own particular and engaging brand of humour. In congratulating Dr. Blount on his concise summary of the present standpoint on poultry meat inspection, Dr. Gordon levelled one serious criticism, namely, that he had covered the subject too completely and left little to discuss. The duty of an opener was, he thought, to be provocative and to stimulate further discussion. The most that he could attempt to do was to pick up a loose thread here and there and possibly to amplify his reference to the public health aspect of egg products as distinct from poultry meat.

As Dr. Blount had pointed out, there was no existing legislation in this country for the inspection of poultry meat with the exception that it was presumably an offence for diseased carcases to be exposed for sale and that an inspector had the right to examine such birds as he considered to be necessary. Unfortunately, as distinct from larger animals, only a small proportion of the table poultry reached the public through recognised channels where such inspection would be available. Since the war, price controls had meant that even fewer carcases than normal were available for inspection.

Dr. Blount had enumerated the common diseases affecting poultry, and he (Dr. Gordon) thought it could be agreed that few were likely to be transmissible to man through poultry meat, particularly in cooked carcases. The question

of the direct transmission to man from handling diseased carcases or through egg products was a different story, and one which he would attempt to deal with later. The problem, therefore, appeared to resolve itself into three points: (1) The wholesomeness of the product for human food, and possibly in that respect the repulsive nature of some diseases was of more importance than the actual danger from their consumption; (2) the danger to the industry by the possible distribution of offal from such infected carcases through swill and other means; (3) (and that applied more to poultry egg products), the possible repercussion on the industry itself should any doubt arise in the minds of the consuming public as to the safety of those products.

With regard to the first point, Dr. Blount had listed the comparative incidence of the common diseases met with at their battery laying trial and compared them with that at the National Laying Trial. It would be seen that those figures resembled one another very closely, but it should be borne in mind that to some extent they differed from the mortality rate experienced on the general farm, since at laying trials the birds were kept under a fairly high degree of isolation and epidemic disease played a small part. It was those infectious diseases which presented the greatest difficulty, since in many cases post-mortem findings were few and seldom characteristic. In view of that, he thought it should be agreed that no birds which had died should be exposed for sale. From fifteen years experience of poultry post mortem service he was still of the opinion that there was nothing more repulsive than the carcase of a dead bird—the pectoral muscles, usually fevered or parboiled in appearance, the peritoneal cavity, often containing an evil-smelling hæmorrhagic or egg-stained exudate, and the vent and head regions usually fly-blown and showing rapid putrefaction.

The difficulty, however, arose regarding culls and birds slaughtered as carriers or in-contacts to disease. That problem was much more serious than in the large animals, where treatment was usually carried out until the bitter end. Poultry farmers, on the other hand, not only killed unthrifty birds, but in the face of an outbreak of disease would rigorously cull for table both the in-contacts and those showing early clinical symptoms. It was in those cases that some sort of guidance was required and they must temper their ruthlessness with common sense. For example, in the blood test for the eradication of pullorum disease or fowl typhoid, it was recognised that reactors to the test were suitable and might, in fact, be used for table purposes. Any condemnation of such reactors for human consumption would undoubtedly have a serious repercussion on that policy.

In the United States, although inspection of poultry meat was not compulsory, most plants processing poultry did apply to the Bureau of Animal Industry for veterinary inspection. The Bureau supplied an inspector, whose salary was paid by the processing firm. The firms using that service could stamp their products as having passed Federal meat inspection, and certain States or cities required such inspection of products which were to be retailed

within their boundaries. Those veterinary inspectors were usually responsible for the general hygiene of the processing plant and the workers. Ante-mortem or post-mortem inspection of poultry or domestic rabbits was made only by an inspector who was a veterinarian, and each carcase or any part thereof which was found to be unwholesome or otherwise unfit for human food, was condemned by the inspector, and received such treatment under the supervision of the inspector as would prevent its use for human food and, in addition, preclude the dissemination of disease through its consumption by other animals. Further, all substances and their ingredients used in the manufacture of any table product were required to be clean, sound, wholesome and fit for human consumption. Those extracts were taken from the U.S. Department of Agriculture "Rules and regulations governing the inspection and certification of dressed poultry and table products thereof."

In the United States, meat inspection of poultry was really limited to the regulations which were enforced by certain municipalities. The main object was the removal of manifestly unwholesome dressed poultry from the local markets; that inspection was limited to a very small percentage of the table poultry poundage which passed through the hands of the few eviscerating and processing plants which maintain a private veterinary meat inspection service under government supervision. There were some seventy plants using that service and the veterinary personnel numbered approximately one hundred (Lynn, 1944). Broadly, poultry showing the following diseases and defects were removed from marketing by that service: tuberculosis, emaciation, septicæmia, leukosis, peritonitis, abscesses, parasites, putrefaction, contusions, cadavers and tumours. To those may be added such lesions as a trained veterinary inspector would naturally select as a cause for condemning meat. The service included sanitary supervision of the plant, its personnel, cleanliness of the inspected carcases before packing, and disposal of rejected material. An excellent description of the method of eviscerating poultry in such processing plants and the associated veterinary inspection was given by Savage (1944).

With regard to tuberculosis, Dr. Blount had stated that modern poultry practices were tending to eliminate tuberculosis; that was very often the case in the specialised flocks, but in his (Dr. Gordon's) experience it was one of the commonest causes of disease in domestic or backyard poultry, where "Henrietta" had become the family pet and kept as long as she was able to walk and eat. Usually the family could not bear the thought of eating Henrietta, and so the local butcher was usually asked to slaughter her for his own use or retail sale. It should be remembered that, in tuberculosis, although macroscopic lesions of the visceral organism were readily detected, probably the commonest site was in the marrow of the long bones, particularly in the stifle and hock joints, and it was reasonable to assume that in those sites the bacilli might resist cooking.

As distinct from pullorum or typhoid testing, it should be ensured that reactors to the tuberculin test were not used for table purposes. In 1941,

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Spears and Dobson showed that a high percentage of reactors showed no macroscopic evidence of tuberculosis, although evidence of infection was found on biological tests of bone marrow and other tissues.

Although tuberculosis has been regarded as rare in water fowl, it was shown (Gordon *et al,* 1941) that in one flock lesions of tuberculosis were found in 25 per cent of the adult birds. In recent years some forty cases of tuberculosis in human beings had been said to be due to the avian bacillus. The danger of infected poultry offal in the transmission of disease to swine through swill had already been stressed by Dr. Blount.

The other diseases which he had listed from the U.S. regulations had been amply dealt with by Dr. Blount, who had, in fact, added quite a number of his own which should be included in the rejects.

To Dr. Blount's list he suggested two possible additions: one was the danger to the human being from the consumption of carcases of poisoned poultry. Poisoning of poultry from phosphorus, zinc phosphide and arsenic, were by no means uncommon, and although most of the carcases would be those of dead birds, and therefore rejected on that ground, he had encountered one case where a sufficiently high mortality so perturbed the farmer that he slaughtered some seventy birds showing clinical symptoms. In that particular case poisoning was due to phosphorus acquired as a result of a smoke bomb which had been liberated during army manœuvres. As the officer in charge was held responsible for the incident, and compensation was paid to the farmer, the advice of the Poultry Research Station was sought as to the safety of the slaughtered carcases for use in the officers' mess.

Zinc phosphide poisoning had been comparatively common in recent years, and since there was some deposition of phosphene in the tissues, it would be advisable that such carcases should be rejected. That would, of course, also apply to arsenical poisoning. With regard to arsenicals, it should be remembered that arsenic preparations were commonly used in the control of blackhead in turkeys, and it was suggested that turkeys so treated should not be used for table purposes until at least fourteen days after inoculation.

Dr. Blount's point regarding the danger to the industry from the possible spread of infection through infected offal had been largely covered; it was, in fact, dealt with in the U.S. regulations, which stated that condemned carcases should receive such treatment under the supervision of the inspector as would preclude dissemination of disease through their consumption by animals. That point was well worth bearing in mind, particularly in view of the high proportion of outbreaks of fowl pest which originated through the consumption of swill containing the offal from infected poultry. In a survey of the first five hundred outbreaks in England, some 33 per cent originated in that way, and there was little doubt that other diseases might be similarly disseminated. Those engaged in meat inspection were of considerable help in identifying many of

the earlier outbreaks; that was particularly the case in the Birmingham area, where a number of the outbreaks were diagnosed and brought to the notice of the official laboratory by the staff of the veterinary and food inspection department in that city.

His final point that the veterinary profession and the industry should play a part in that problem was best illustrated by the present position regarding avian salmonella infection and human food poisoning.

There had been numerous references in the past, particularly in the annual reports of medical officers of health, of cases of fatal poisoning in man from the consumption of infected duck eggs, and in 1948, Gordon and Buxton surveyed the literature and reported a fatal case in which the same organism was recovered from the blood and excreta of the patient and from eggs laid by the supplying flock and from the ovaries of the ducks which had laid the infected eggs.

At the eighth World Poultry Congress in Copenhagen, Goresline drew attention to the increasing importance of salmonella organisms in food products —particularly poultry and egg products—due to the increased number of reports of salmonella food poisoning in man. Some sixty of the one hundred and fifty of the total of salmonella types had been isolated from the avian species, and of those at least fifty-six had been found in human beings; while some fifty-two strains had been isolated from dried eggs. Since only an occasional egg was contaminated with salmonella organisms, only a small number of persons could be infected at one time when shell eggs were consumed. In the liquid, frozen and dried egg processing, however, large numbers of eggs were mixed in the same vat, and one contaminated egg might be responsible for infecting an entire consignment of the processed material, and each individual person consuming that material or preparations made from it would receive an equal potential inoculum. The Medical Research Council had stated that salmonella organisms were found in 16 per cent of some one thousand routine samples examined; while Solowey said 35 per cent of over five thousand samples of died egg powder were positive for salmonella.

The problem was increased when one realised that in man salmonellosis was mainly an infection of children and invalids, and that the materials prepared from such products as slightly-cooked foods, meringues, cream-bun fillings, egg drinks, ice-cream and the like were products most favoured by children. There was little doubt that the main source of contamination of those egg products arose from the infected hens themselves, and since the eradication of such ubiquitous organisms from poultry flocks was, at the moment, impracticable, the solution must to a large extent lie in the hands of the egg producers and processers. It had been shown that, in salmonella infections, survivors of an outbreak harboured the organism in the bowel and that the eggs became contaminated on the outside of the shell with infected fæcal material at, or after, laying. Experiments had indicated that improved hygiene and egg pro-

duction did, to a large extent, reduce shell contamination. The wiping of dirty eggs was contra-indicated, since it spread the area of inoculum and hastened the rate of penetration, and the cloth and water rapidly became heavily contaminated.

It was shown (Solowey) that 5 per cent of egg samples from clean eggs were positive to salmonella, 15 per cent of those from light dirty eggs, and 20 per cent of those from washed dirty eggs; while Canter and MacFarlane had shown that salmonella infection was more commonly found in low-grade and cracked eggs than in high-grade. That suggested that one method of reducing the incidence of salmonella would be to use for marketing or for processing only clean eggs of high grade. In this country there appeared to be no sanitary legislation regarding egg-packing stations; the onus for producing clean eggs was on the egg supplier and not on the packer. Grading was in quality only, and dirty and cracked eggs were graded as seconds, and, he believed, distributed by the Ministry of Food as " off-ration " eggs.

There was no control of the cleaning, which was carried out on the producer's premises, and usually that was done by wiping or washing in dirty water. It would appear to be preferable that that should be done centrally under standardised conditions and supervision at the packing station.

Work had been carried out on the use of detergents for the cleaning of eggs with the quaternary ammonium chloride type, and with numerous germicidal solutions, in an attempt to sterilise the egg shell. Some machines were on the market for that purpose. It had also been demonstrated by Winter *et al.* that salmonella could be destroyed by pasteurisation of the liquid egg, and Funk had shown that if shell eggs were subjected to certain temperatures, bacteria in the interior of the egg were killed.

The problem of salmonella in poultry meat was not so acute as that in eggs, and contamination of the body surface was mainly due to insanitary methods of handling. The preparation of viscerated and cut up poultry products increased the chance of salmonella contamination. Hinshaw had also suggested that poultry might acquire infection from carrier attendants, and vice versa, and where the danger was probably more likely to occur was in the hatcheries.

The problem of salmonella infections in public health had received a great deal of attention on the continent, and there was legislation covering the sale of duck eggs which required that they be stamped and that they be boiled for ten minutes before use. In certain countries ducks could not be kept on premises where food products were sold or prepared. There was no doubt that that problem should receive the attention of the veterinary professsion and the poultry industry, in whose hands mainly lie the most effective methods for control of those infections. Such efforts would not only reduce the mortality in chick rearing, but would do much to increase the consuming public's confidence in poultry products.

REFERENCES

Cantor, A., McFarlane, V. H. (1948): Quoted by Goresline, H.
Gordon, R. F., Garside, J. S., Dobson, N., and Reid, J. (1941): *Vet. Rec., 53,* 40, 575-580.
Gordon, R. F., and Buxton, A. (1945): *Bull. Min. Hlth.,* E.P.H.L.S., *4,* 46.
Goresline, H. G. (1948): *Proc. 8th World Poultry Congress, 1,* 604.
Hinshaw, W. R., and McNeil, E. (1948): *Proc. 8th World Poultry Congress, 1,* 599.
Lynn, E. M. (1944): *J. Amer. Vet. Med. Ass., 104,* 206.
Spec. Rep. Ser., Med. Res. Counc. (1947), 260. London.
Savage, R. E. (1944): *J. Amer. Vet. Med. Ass., 104,* 206.
Solowey, M., Spaulding, E. H., and Goresline, H. (1946): *Food Research, 53,* 380.
Spears, H. N., and Dobson, N. (1941): *Vet. Rec., 53,* 26, 365-370.
Winter *et al.* (1946): *Amer. Jour. Pub. Hlth., 36,* 5, 451.

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FELINE ENTERITIS IN CAPTIVE LEOPARDS IN CEYLON

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THE OBJECT of this communication is to record the existence of feline enteritis in captive leopards in Ceylon and also some preliminary transmission and immunisation experiments.

This virus infection of Felidæ has been described and investigated in England by Kirk (1925), Hindle and Findlay (1932), Dalling (1935), Cockburn (1947), Jennings (1949), Montgomerie and McIntyre (1949); in France by Verge and Cristoforoni (1928), and Urbain (1932); in the U.S.A. by Leasure, Lienhardt and Taberner (1934), Lawrence and Syverton (1938), Hammon and Enders (1939), Salsbery (1939), Arlein (1940), Riser (1947), Goss (1948); in Brazil by Macchiavello and Coutinho (1940), Silva (1940), Torres (1941). Although cross-immunity experiments, using strains of virus from the different countries, have not been recorded, it appears probable that feline enteritis of British authors is identical with the "Infectious Feline Agranulocytosis" of Lawrence and Syverton, and of the "Malignant Pan-leucopenia of Cats" of Hammon and Enders (Jennings, 1949). Similarly, there seems no doubt but that the disease in captive wild Felidæ described by Hindle and Findlay (1932), Cockburn (1947) in England, Torres (1941) in Brazil, and by Grosso and Rodriguez (1948) in Argentine was feline enteritis. Cockburn (1947) records the disease in all the captive Felidæ in Regents Park Zoological Gardens except the lion. Goss (1948) states that the disease has been seen in all the wild Felidæ kept in the Zoological Park of New York with the exception of the lion. He also states that the only animal outside the Felidæ in which the virus has been demonstrated is the racoon, a member of the family *procyonidæ*; incidentally, according to this author, this animal is also susceptible to the virus of canine distemper.

In the Zoological Gardens, Dehiwela, near Colombo, the disease has been seen only in leopards (*Panthera pardus*), although lions, tigers, panthers and jaguars as well as numerous other carnivores, including civets and mongooses, have been exposed to infection. The most likely explanation is that the leopards are captured in the Ceylon jungles as cubs usually only a few weeks old and brought to the Gardens. Thus they are exposed at a susceptible age to infection from the numerous semi-wild mongrel cats which prowl round the cages at night. Feline enteritis is a common disease of domestic cats in Colombo, but appears to be highly fatal only to the more sheltered pure breeds such as Siamese and Persians. The present writer has found that the adult mongrel cat is strongly immune and that only an occasional mongrel kitten is susceptible. This resistance of ordinary cats has handicapped transmission work.

The records at the Zoological Gardens show that every year several young leopards have died from enteritis. The symptoms are well known to the veterinary officers, the superintendent and his assistants, viz., anorexia, vomition of glairy brown fluid, intense depression; evidence of abdominal pain sometimes is so severe as to cause maniacal symptoms. Death takes place within 48 to 72 hours from the onset. Diarrhœa, as has been remarked by Kirk (1925) in his description of the disease in domestic cats, is seldom seen.

The first case which was examined was a young leopard about 4 to 5 months old; two other young leopards had died a few days earlier having shown the characteristic signs. This third animal was moribund when seen and was autopsied soon after death. The post-mortem findings were severe enteritis of the small intestine and less pronounced inflammation of the colon and rectum; the lymph nodes of the mesentery and bronchi were swollen, the liver, spleen, kidneys, lungs were apparently normal. There was infestation with hookworms (*ancylostoma caninum*). At the same time there was in contact with these three young leopards an older female leopard; this animal was thought to be ill, but it did not exhibit the typical signs of vomition and depression and was apparently in fairly good health a few days later. Its subsequent history is the subject of a separate communication.

The second case occurred a week later in a young leopard which had been reared in a private bungalow in Colombo. The symptoms were the usual ones characteristic of the disease, viz., sudden anorexia, vomition of glairy fluid, intense depression, intense abdominal pain. The cub died within 48 hours of the onset; according to the lady who had reared it, it was in extreme agony before death. On post-mortem examination made soon after death, very severe hæmorrhagic inflammation of the colon and rectum and less intense inflammation of the small intestine were found. The lymph nodes of the abdominal cavity were swollen; the other tissues appeared normal. Despite a careful search, no evidence of hookworm or other helminths was found. The liver and spleen were removed and minced as aseptically as possible; a portion was placed in 50 per cent glycerol saline, the remainder was ground with sterile sand and suspended in physiological saline to make approximately a 10 per cent suspension. This suspension was formalinised to 0.2 per cent and kept at 50 deg. C.

for seven days. It was then tested for bacteria by culture and for non-pathogenicity to cats by inoculation of kittens. After another seven days at 50 deg. C. it was inoculated in doses of 5 c.c. into each of five young leopards brought into the Zoological Gardens to replace those which had died. No untoward reactions were noted and a second dose of 10 c.c. was injected after ten days. Two young black panther cubs also were injected with 5 c.c. of suspension. All these animals have remained healthy up to the present time, over five months since inoculation. Despite the absence of controls, the superintendent of the Gardens is convinced of the efficacy of the vaccine in conferring at least a temporary immunity. It is intended to pursue investigations on the virus and on immunisation when a supply of susceptible cats is available. Here it should be mentioned that immunisation of cats by means of formalinised suspension of tissues was carried out by Urbain (1933), Leasure and others (1934), Dalling (1935), Salsbery (1939), Grosso and Rodriguez (1948); Kirk (1948) states that he and other veterinary practitioners in England had gratifying results with the vaccine and serum prepared by the Wellcome Research Laboratories before 1939 and he regrets the fact that these products are not now available. Montgomerie and McIntyre (1949) have immunised captive wild Felidæ with formalinised spleen suspension.

Attempts to transmit the infection to mongrel cats have met with only limited success. Using spleen and liver saline extract from leopard I as inoculum, two young cats were inoculated, one subcutaneously (cat 1), the other by intra-nasal instillation (cat 2). The first cat survived for 15 days, cat 2 showed slight rise in temperature and died within seven days; on post-mortem examination lesions of enteritis were found. A saline extract of the liver and spleen was inoculated subcutaneously into cats 3 and 4. Cat 3 died three days later, having shown no rise in temperature nor any significant fall in leucocyte count. Lesions of acute enteritis were found on post-mortem examination. Cat 4 developed an abscess at the site of inoculation; the leucocyte count rose from 18,000 per cub. mm. on the third day after inoculation to 52,000 per cub. mm. After opening of the abscess, the leucocyte count fell to 28,000 per cub. mm., but rose gradually to 91,000 per cub. mm. just before death. The causal organism was seen to be a streptococcus, but no cultures were made. Saline extract of liver and spleen from cat 3 was instilled intra-nasally into cats 6, 7 and 8. The results with these animals were inconclusive; cat 7 died on the fifth day and cat 8 on the sixth day after inoculation. In cat 7 there was slight enteritis; in cat 8 there was severe hæmorrhagic enteritis and also patchy congestion of the lungs. The temperature of cat 7 had risen to 102.6 deg. F. by the third day, but fell again to normal before death; in cat 8 the temperature remained within normal range. Similarly in cat 6, there were no significant rises in temperature nor decrease in leucocyte count; this animal survived for 17 days and was then chloroformed; no significant lesions were found on post-mortem examination.

Using liver and spleen extract from leopard II, more definite results were obtained. In the first set of transmission experiments, the extract, which was made from liver and spleen kept at 5 deg. C. in 20 per cent glycerol saline for

only two days, was instilled intra-nasally into three cats. Two of these cats were adults and did not show any signs of illness beyond a slight rise in temperature on the fourth, fifth, sixth, seventh and eighth days after instillation. On the sixth, seventh, eighth and ninth days there was a definite fall in the leucocyte count which was still within the normal range. The third cat, a younger animal, showed a gradual rise in temperature up to the sixth day when it was 105 deg. F., remaining at this point on the sixth day; death occurred in the night of the sixth to seventh day. The erythrocyte count did not alter much, falling from over six million per cub. mm. to just below five million per cub. mm. The leucocyte count fell from 33,000 per cub. mm. on the day of infection to 18,000 on the third day, then to 13,000 on the fourth day, 6,400 on the fifth day, approximately 1,000 on the sixth and seventh day, and 600 a few hours before death. On post-mortem examination of this cat, acute inflammation of the cæcum and large colon was found; the liver, spleen and kidneys were normal in appearance, but several patches of consolidation in the lungs were found.

In view of the difficulty in obtaining susceptible cats, it was decided to postpone further transmission experiments. The glycerolated minced liver and spleen from leopard II was kept at 5 deg. C. for 18 weeks and then used to make a 10 per cent saline extract which was inoculated subcutaneously into two kittens and instilled intra-nasally into three kittens. The results obtained with these five kittens were definite and reasonably uniform. One of the two inoculated subcutaneously showed no rise in temperature for five days; unfortunately on the sixth day, being Sunday, the temperature was not taken and the animal was found dead on the morning of the seventh day. However, the animal attendant stated that the cat had appeared to be depressed and had vomited. In the other kitten inoculated subcutaneously, the temperature rose to 103 deg. F. on the second day after injection, then fell to 100.4 deg. F. by the fourth day and rose again to 105.6 deg. F. on the seventh day. The leucocyte count was 5,200 per cub. mm. on the fifth day and 4,000 per cub. mm. on the seventh day. The animal was showing signs of abdominal pain, the mucous membranse of the anus was inflamed, there was diarrhœa and vomiting of frothy bile-stained fluid.

In the three kittens infected by intra-nasal instillation, there was no significant rise in temperature for five days; again, unfortunately, the temperature was not taken on the sixth day. On the seventh day the temperatures were 98.6 deg. F., 102.2 deg. F. and 102.4 deg. F. respectively; all three cats were off their food, depressed and vomiting frothy, bile-stained fluid. There was no evidence of diarrhœa. The leucocyte counts had fallen to 600, 1,200 and 600 per cub. mm. respectively on the seventh day. All four surviving cats were chloroformed and autopsied.

The post-mortem findings in all five cats were more or less similar, viz., slight excess of fluid in the peritoneal cavity, slight inflammation of the small intestine, swelling of the lymph nodes, especially of those near the ileo-cæcal junction; stomach empty except for a little bile-stained frothy fluid; liver, spleen, kidneys, lungs, heart apparently normal.

Remarks

For various reasons, chiefly because of the lack of an adequate supply of susceptible cats, it has not been possible to carry out transmission experiments using filtrates. There seems to be no doubt that most of the adult mongrel cats of Colombo are strongly resistant. The writer has been informed by Mr. L. A. P. Britto-Babapulle, B.Sc., M.R.C.V.S., that in Colombo feline enteritis is seasonal in its occurrence, appearing in epizootic form in the months of May, June and July. The disease in the leopard was seen during those months.

It is intended to breed pure-bred Siamese cats in isolation for further investigation.

Summary

(1) A disease of leopard cubs in captivity in Ceylon has been described.

(2) The signs and lesions resembled those of feline enteritis described in domestic cats and captive Felidæ in other countries.

(3) The disease was reproduced in young mongrel cats by subcutaneous inoculation and intra-nasal instillation of an unfiltered saline extract made from the liver and spleen minced and preserved at 5 deg. C. for 18 weeks.

(4) In the experimentally infected cats, rise in temperature was usually only slight; the characteristic signs were anorexia, intense depression and vomiting of frothy bile-stained fluid. Diarrhœa was unusual. Leucopenia was marked.

(5) Experiments in immunisation have not been possible, but five young leopards and two young black panthers were injected with a formalinised saline extract of liver and spleen; although undoubtedly exposed to natural infection, these animals have remained healthy over a period of five months.

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REFERENCES

Arlein, M. S. (1940): *Nth. Am. Vet., 21.* Quoted by Kirk (1947).

Cockburn, A. (1947): *Vet. Jl., 103,* 261.

Dalling, T. (1935): *Vet. Rec., 15,* 283.

Enders, J. F., and Hammon, W. D. (1940): *Proc. Soc. Exp. Biol. and Med., 43,* 194. Quoted by Jennings.

Goss, L. J. (1948): *Am. J. Vet. Res., 9,* 65.

Grosso, A. M., and Rodriguez, A. (1948): *Gac. Vet. B. Aires, 10,* 51-60. Abstract in *Vet. Bull., 19,* 276.

XUM

Hammon, W. D. (1940) : *Anat. Rec., 76,* 259.
Id. (1940) : *Proc. Soc. Exp. Biol. and Med., 43.* Quoted by Kirk (1947).
Hammon, W. D., and Enders, J. F. (1939) : *J. Exp. Med., 69,* 327.
Id. ibid., 70, 557.
Hindle, E., and Findlay, G. M. (1932) : *J. Comp. Path., 45,* 11.
Id. (1933) : *Proc. Roy. Soc. Med., 26,* 197.
Jennings, A. R. (1949) : *Brit. Vet. J., 105,* 89.
Id. (1949) : *Vet. Rec.*
Kirk, H. (1925) : "Diseases of the Cat."
Id. (1933) : *Discussion Proc. Roy. Soc. Med., 26,* 203.
Id. (1947) : "Index of Diagnosis." Baillière, Tindall and Cox, London.
Lawrence, J. S., and Syverton, J. T. (1938) : *Proc. Soc. Exp. Biol. and Med., 38,* 914.
Id. et al. (1940) : *Am. J. Path., 16,* 333.
Id. (1940) : *J. Bact., 39.*
Leasure, E. E., Lienhardt, H. F., and Taberner, F. R. (1934) : *Nth. Am. Vet., 15,* 30. Quoted by Stein (1942).
Macchiavello, A., and Coutinho, A. B. (1940) : *Brasil. Med., 54,* 113. Quoted by Stein (1942).
Montgomerie, R. F. (1949) : Private communication.
Riser, W. H. (1947) : *Am. J. Vet. Res., 8,* 82.
Salsbery, C. E. (1939) : *J. Am. Vet. Med. Ass., 95,* 195.
Silva, M. Jr. (1940) : *Hosp. Rio de J., 18,* 1015. Abstract in *Vet. Bull., 19,* 102.
Stein, C. D. (1942) : "Keeping Live Stock Healthy." U.S. Government Printing Office.
Syverton *et al.* (1943) : *J. Exp. Med., 77,* 41. Quoted by Jennings (1949).
Torres, S. (1941) : *Nth. Am. Vet., 22,* 297. Quoted by Stein (1942).
Urbain, A. (1933) : *Ann. Inst. Pasteur, 51,* 202. Quoted by Stein (1942).
Verge, J., and Cristoforoni, N. (1928) : *C.R. Soc. Biol., 99,* 312.

THE CAMEL IN ANCIENT EGYPT

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Introduction

ALTHOUGH the camel was believed to be amongst the animals used in Ancient Egypt for transport and food consumption for thousands of years, yet no reference to this has been mentioned in ancient history. It could not even be traced in any of the writings on the Egyptian monuments which usually explained the life, customs and existing creatures of that ancient time. This was an outstanding excuse for the absence of knowledge of Egyptologists as far as the camel is concerned.

Biology and Evolution

North America is considered to be the land in which the first known camel originated. This was at the Tartiaer period (about one million years ago). The camel migrated towards two directions, one to Asia and the other to South America, when the family camelidæ was abolished from North America.

At present there are two distinct species of the *Genus camelus,* viz., the Bacterian (two-humped camel) and the Arabian (one-humped dromedary). The dromedary is common in Arabia and Africa, but the Bacterian camel, although

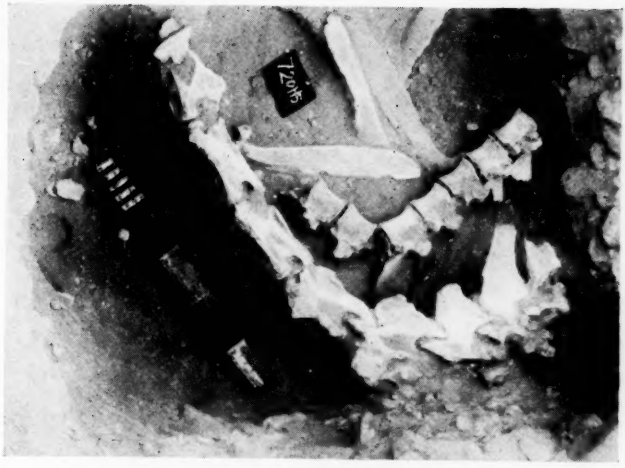

FIG. 1.

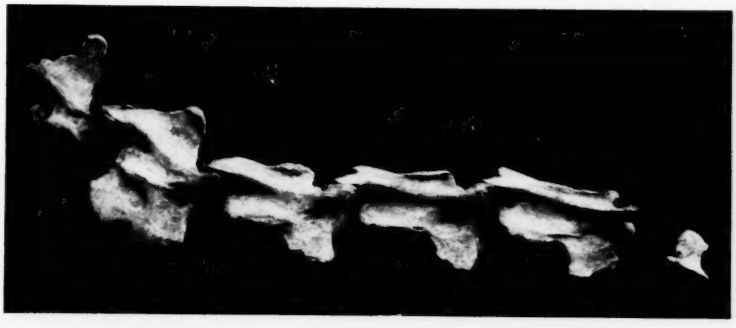

FIG. 2.

FIG. 3.

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FIG. 4.

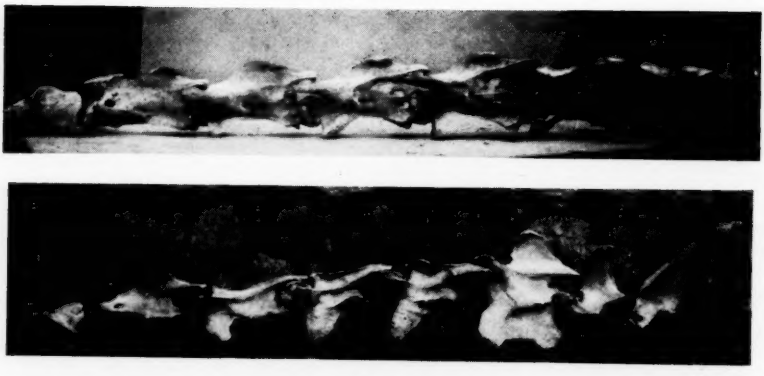

FIG. 5.

(*Article by N. George, page 76*)

common in Egypt in the ninth and tenth centuries of our era, is regarded now as an inhabitant of Asia.

Material

On April 26, 1947, Mr. Z. Y. Saad, the Director of H.M. King Farouk's excavations at Helwan, near Cairo, came across an intact tomb. It was given the number 720 of the fifth season. The tomb was found rectangular in form, cut in gravel, and measuring 95 cms. from north to south, 75 cms. from east to west, and 110 cms. deep. The sides are without any buildings. At the bottom, lying on the floor, were some bones of the neck of a big animal, and close to them some large ribs, which belong to the same animal (Fig. No. 1). Mr. Z. Y. Saad stated that the age of these tombs was 3200 B.C.

On November 22, 1947, I was delegated by Fouad I University to examine these bones for identification.

The bones, after being preserved by the specialist, were submitted to me for examination. They were found to be the third, fourth, fifth, sixth, and seventh cervical vertebræ and the odontoid process of the second cervical vertebræ of a camel (Fig. No. 2), also the first seven thoracic vertebræ (Fig. No. 3) and eight ribs of the same animal (Fig. No. 4).

The excavator thought that these bones would belong to the Giraffe, as he and nearly all those interested in such excavations had never heard of any such bones of a camel being found in connection with the monuments of Ancient Egypt. A detailed comparison between the bones of the two animals was undertaken.

Leese (1927) stated that, in the normal standing position, the cervical vertebræ in the camel are arranged in a semicircular curve, with the concavity uppermost, and that the great length of the neck is not attained by any increase in number of cervical vertebræ above that common to most mammals, but by the much greater length of each individual bone.

The atlas, or first, cervical vertebra has small wings, which can be felt easily through the skin in the living animal; the cavities which articulate with the occipital bone are deep. The axis, or second, cervical vertebra is elongated and narrow, and its neural spine is low; the inferior spine is insignificant.

The other five cervical vertebræ are long. The anterior articular surfaces of their bodies are less convex and posterior ones are less concave than are those of the horse, and this, along with its well-developed intervertebral discs, gives great freedom of lateral movement between the bones of the camel's neck. The intervertebral notches are deep. The neural spine is low in the fifth vertebra, tall in the sixth and seventh. Each transverse process has two divisions, the anterior one being always the largest and pointing in a downward direction, and, being flattened from side to side, gives protection to the important organs and vessels which run down the convexity of the inferior surface of the neck. This anterior division is of great size in the fifth vertebra, in which

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it projects downwards in a flattened plate which is equally as long as the body of the vertebra itself. The lower ends of these anterior divisions can be grasped between the thumb and finger through the skin in the living animal. The post divisions of the transverse processes of the third to seventh cervical vertebræ form little prominences at equal intervals down the middle of the side of the neck.

The transverse process of the seventh cervical vertebra is much smaller than those of the anterior bones.

Chauveaus (1891) stated that the cervical vertebræ in the camel are longer than in the other large domestic animals, and that they form one-third of the total length of the spine. Their vertebral laminæ are deeply notched before and behind, and allows them easily to enter the spinal canal. Their articular processes are convex, and that from the second to the sixth, inclusively, the vertebral foramen is small and deeply placed in the laminæ.

The atlas is characterised by the absence of the inferior tubercle and the shortness of the transverse process, which have thin and sharp borders. The vertebral foramen makes a somewhat long course in its transverse process and opens at the bottom of the excavation, in which it meets the anterior intervertebral foramina, and that the latter is double.

The axis is very long and constricted in the middle, and the inferior crest is only slightly marked. The intervertebral foramina are double and the largest is divided by a bony septum; the odontoid process is as in the ox, and the other cervical vertebræ gradually diminish in size and increase in thickness from before to behind. In the third, fourth and fifth the transverse processes are bi-tuberculated, while in the sixth the transverse process is a wide and thick plate, inclining downwards. The seventh is recognised by its long, spinous process and the smallness of its transverse process, which has a larger and more obvious vertebral foramen than the other vertebræ.

Abdel Aziz Nomani (1911) stated that the camel presents seven long but narrow cervical vertebræ, forming one-third of the total length of the vertebral column. The anterior and posterior notches are deep and the articular processes are convex and small. With the exception of the seventh, the vertebral foramen cannot be seen from the outer aspect. Each vertebral foramen is situated at the anterior aspect of the root of the anterior oblique processes and opens in the middle of the neural ring towards its pedicles.

The atlas, with its long axis, is almost perpendicular in the living subject, but it is small, with a long and narrow neural canal; for the purpose of comparison, it is assumed that the bone is placed horizontally with its wings situated in an oblique direction downwards and forwards, and that each wing has a thing edge ending inferiorly with a small tubercle.

The inferior tubercle is absent, and between the ring and the wing there is a large foramen beginning from under the latter and opening posteriorly on its side. This takes the place of the posterior foramen. The antero-internal

and antero-external foramina are present, and from the bottom of the furrow connecting the two a third small foramen leads to the neural ring. The neural spine is not so prominent and the facets on the posterior edge are markedly concave.

The axis is very long and constricted in the middle, and the posterior oblique processes are smaller than in the horse. The superior spine forms a long, thin and prominent ridge, ending posteriorly in a bifid tubercle for ligamentous attachment. The anterior intervertebral notch is double, and the larger of the two is divided by a long septum. The odontoid process is shorter, less pointed, and more spout-like dorsally.

The neural ring is very long and narrow; the transverse processes are single but larger, and slightly more curved than in the horse. The posterior end of the body is nearly flat, while the anterior notch is not large, as in the horse.

The third, fourth and fifth cervical vertebræ are characterised by the greater length and size of their bodies, and each of their transverse processes is divided into two parts, a broad, descending plate and a horizontal plate. The posterior end of their bodies is flat, while the anterior is markedly convex.

The bodies of these vertebræ increase in width from the second to the sixth, but decrease in length from the second to the last, and inferior spines diminish from the third to the seventh, and each spine shows two small nodules posteriorly, but the transverse processes increase from the third to the seventh. The superior spines are higher than both horse and ox, while the inferior surface is wide and concave and its spine resembles that of the vertebra dentata.

The sixth cervical vertebra is very large and massive and it carries a trifid transverse process, which is composed of two large descending plates and one small horizontal plate inclined backwards, and the superior spine is well marked, while the inferior is very faintly marked.

The seventh cervical vertebra is shorter and larger than that of both horse and ox and its neural spine resembles that of a lumber vertebra, having a square-cut end and a forward inclination. The inferior spine is represented by a slight ridge and the vertebral foramen is large, but is absent sometimes. The transverse process is double and relatively small, the facets on articular processes are large, the neural ring is short and nearly triangular, the posterior end of the body is concave, the inferior spine is faint, and the body is very broad.

The posterior facets of the body are large and more concave for the head of the first rib.

The Cervical Vertebræ in the Giraffe (Fig. No. 5)

No books on the description of the bones of the Giraffe could be traced at present; the skeleton of the Giraffe which stands in the museum of the Cairo Zoo Gardens was taken as a reference.

These are seven in number and are arranged in a straight line upwards and forwards between the thoracic region and the head.

When compared with those of the camel, they presented the following differences:—

(1) The bodies of the vertebræ from the first to the last are longer, narrower, and somewhat constricted in the middle.

(2) The anterior ends of the bodies are markedly convex and the posterior is markedly concave. The convexity is triangular in shape and the base upwards.

(3) *The transverse process* is plate-like and directed downwards and forwards. It is continued backwards by a sharp, concave edge which reaches the posterior extremity.

(4) *The inferior spinous process* is represented by a faint ridge anteriorly and absent posteriorly.

(5) *The neural canal* has a triangular anterior end with its base upwards, while posteriorly the base is downwards. The floor is markedly concave, being boat-shaped.

(6) *The superior spinous processeses* from the third to the sixth are well developed, nearly equal in length; each subsides when traced forwards and backwards. Each presents a grooved, rough summit.

(7) *The seventh cervical* presents a pyramidal-shaped superior spinous process, somewhat rough and not grooved.

(8) *The anterior oblique process* is continued backwards and downwards by a ridge which reaches the lower third of the posterior extremity. This ridge is perforated by the vertebral foramen (Fig. No. 5).

(9) *The vertebral foramina* increase in width and length from the third to the sixth. In the seventh vertebra it is much wider and shorter.

(10) *Atlas*:—

(1) It is longer and constricted in the middle.

(2) The wings are represented by two narrow transverse processes which form small, pointed processes, which project backwards and inwards behind the posterior articular facets.

(3) The neural spine is represented by a very large, rounded tubercle and the inferior spine is represented by a thick ridge, terminating behind by a large, rough, rounded tubercle.

(4) The arch shows superiorly a deep, wide notch on its posterior aspects.

(11) *Axis*:—

(1) The anterior aspect presents a well-marked odontoid process which is spoon-shaped, being wide, deep and circular.

(2) The neural spine presents a well-marked, beak-like anterior, and (Fig. No. 5) which overhangs the odontoid process and nearly on the same level. This greatly resembles that of the dog. The vertebral foramen is markedly narrow and short, and lies on a lower level when compared with the rest of the vertebral foramina.

MUNICIPALITY OF SINGAPORE

APPOINTMENT OF

DEPUTY MUNICIPAL VETERINARY SURGEON

The Municipal Commissioners of the town of Singapore invite applications for the appointment of Deputy Municipal Veterinary Surgeon. Applicants should be Members of the Royal College of Veterinary Surgeons.

The work involved is mainly clinical and entails assistance in the Animal Infirmary, the Municipal Abattoirs, duties connected with the Prevention of Cruelty to Animals Ordinance and with the Registration and Control of Dogs in the Municipal area of Singapore. The appointment offers a wide range of practical Veterinary experience in all species of animals though mainly in horses and small animals.

The commencing salary offered is from $560 to $660 a month according to age and experience, rising by increments of $30 p.m. (generally) to $1000 p.m. In addition an Expatriation Allowance is payable commencing at $110 p.m. and rising to $190 on $1000 p.m. A variable Cost of Living Allowance is also payable which on $560 p.m. would at present be $192 p.m. for a single man and considerably greater for a married man with children. The Singapore dollar equals 2s. 4d. sterling. Commencing salary, etc. for a single man would therefore be from £1026 to £1420 per annum. The appointment would in the first place be for three years, with annual increments.

A free passage will be provided for the officer and if married for wife and children under 17 years up to a maxima of three full passages. Half pay on voyage. Partly furnished quarters provided if available at 8% of officer's salary; if not available an allowance in lieu. Contributory Provident Fund, full pay leave after four years service, local income tax less than U.K. Medical examination essential.

Applications in duplicate stating age, place of birth, details of education, qualifications and experience, should be lodged with

Messrs. Peirce & Williams (Agents to the Municipal Commissioners)
1 Victoria Street, Westminster, London, S.W.1

not later than March 6, 1950.

Conclusion

The presence of some bones of the camel in the Royal excavations at Helwan, near Cairo, proved almost conclusively the presence of the camel in Egypt during the pre-dynastic period, and appeared entirely from the Third, or possibly the Sixth, Dynasty. This is the view expressed by Zippelius and modern Egyptologists.

The camel being absent from the drawings of the Ancient Egyptian monuments is probably due to the belief that this animal was an evil sign, as it was the only means of transport for the enemies who used to invade Egypt at that time.

REFERENCES

Leese (1927): "The One-humped Camel," page 20.
Chauveaus (1891): "Chauveaus's Comparative Anatomy," page 31.
Abdel Aziz Nomani (1911): "The Comparative Osteology of the Camel," pages 32, 33, 34 and 35.

CLINICAL ARTICLE

LOSSES IN GEESE, DUCKS AND POULTRY, Caused by a Toxin in the Gut Contents which Resembled the Toxin Produced by the Anærobe *C. BOTULINUM*

By O. V. GUNNING, D.V.M., F.R.C.V.S.

DURING the war years, August, 1944, a request was received to examine and investigate a fatal illness in some geese.

On arrival, thirteen geese, four adult ducks and six ducklings were found lying dead. Some were dead when the owner arrived on his premises in the morning. The remainder had died during the day. The dead geese were of all ages. Two geese and two ducks were unaffected, and at this visit no poultry were affected.

The birds had the run of a field where there was a plentiful supply of grass, and they were able to find most of their own living. They all, however, received an additional ration of balancer meal, which was fed in conjunction with cooked kitchen waste which the owner collected from a neighbouring town. The kitchen waste was brought, and kept in, sealed containers.

The affected birds appeared to become suddenly affected, and would fall over in a coma, with general paralysis and inco-ordination. It was noticed that they could remain in this comatose condition for several hours without actually dying.

Carcases of both geese and ducks were submitted to the Institute of Animal Pathology Cambridge, which reported the presence in the gut contents of a

toxin which resembled the toxin produced by the anærobe *C. botulinum*, although they were unable to prove that it was, in fact, this toxin. The report further stated that the tissues of both the goose and the duck were heavily invaded with anærobes post-mortem, which is rather an unusual feature in birds, suggesting that some of their food was undergoing decomposition by anærobes.

It was later ascertained that similar losses had occurred in other geese fed from kitchen waste from the same source. Also, the owner's poultry, which at first were unaffected, later succumbed to the same condition.

Acknowledgment

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